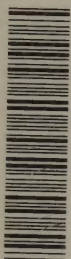


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Brethren, knowing the time, that it is now high time to awake out of sleep . . . the night is far spent, the day is at hand; let us therefore cast off the works of darkness, and let us put on the armour of light. Let us walk honestly, as in day, . . . not in contention and strife. (From Epistle of St. Paul, Rom. xiii. 11-13.)

INTESTINAL TOXEMIA

(AUTOINTOXICATION)

BIOLOGICALLY CONSIDERED

BY

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In Memoriam

DEDICATED TO THE MEMORY OF MY DEPARTED FRIENDS,
COUNSELLORS, AND TEACHERS IN MEDICINE, NOTABLE AMONG
WHOM HAVE BEEN :

Dr. Herman M. Biggs
Dr. David Bouvaird
Dr. William T. Bull
Dr. Francis Delafield
Dr. Austin Flint
Dr. Charles L. Gibson
Dr. Christian A. Herter
Dr. Edward G. Janeway
Dr. Alfred Lee Loomis
Dr. Charles McBurney

PREFACE

One writes on a medical topic for various reasons, rarely, however, in an apologetic way. On a number of occasions what savored of personal criticism has been aimed at me by my colleagues in medicine, especially by gastro-enterologists and internists, for being inactive along literary lines concerning the methods I have employed for years in the handling of the intestinal toxemias. True, such criticism usually wore the friendly guise of a desire for information that would help them in their cases, a noble and sufficient cause for any criticism, although at times this proper basis seemed lacking or questionable.

Beginning with the researches and studies on this subject in 1899, no article of mine appeared on any phase of it until 1910, and after that date none was written for publication up to the time of the first edition of my text-book on "Diseases and Disorders of the Intestines" in 1920. The article (*Medical Record*, Sept. 24, 1910) drew attention to only a limited part of the work, and yet on many occasions before the volume on the intestines appeared it had been brought to my attention that my method was stated to have been employed in this or that case and was being employed by this or that physician or even lay person. In 1910 the studies and applications were developed in many ways beyond what this simple article suggested and since then have broadened tremendously. Therefore it is plain that if only the method suggested up to that time was employed, it was unfair to both the patient and myself to designate it as a method bearing my name. The only reasons I can see for doing so, are either ignorance as to the methods that were being employed or a desire for gain on the part of others. The ignorance I condone and regret, but for the commercial attitude there can be only censure.

Other men have criticized my beliefs in the work, the methods of diagnosis and those of treatment, on the simple

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basis of not knowing anything about such methods and, therefore, they were to be doubted and disparaged. Some have pursued other lines differing in kind and type in the handling of these cases, and their interest in their own methods has brought criticism of mine. But for those who desired to engage in this field, the first and subsequent editions of my work on diseases of the intestines covered it completely enough and widely enough for some degree of satisfaction. In this textbook, matter pertaining to the subject which was entirely technical was omitted, the work being of large size without this, and most of which more properly belonged in works on clinical and laboratory medicine anyway. Enough pertaining to diagnosis and fair types of general culture vaccine methods were presented. That some of the readers were limited in understanding and experience in laboratory procedures and would not undertake to accomplish such or have it done, and had only limited knowledge of immunology, vaccine therapy, etc., were not good reasons for stating that I had not presented the subject in ways understandable to them.

For these, and other reasons, and because I had decreed that the final report of the work had to cover a large number of cases in which constant research and laboratory detail had to be engaged in—work, which at best is far from easy and plain—I concluded not to write this experience in more detail until five thousand cases had been treated. This has taken thirty years of time, constant researching, examinations, checking and rechecking, and the estimations of results immediately accomplished, but, more particularly, results apparent years after termination of treatment. Perhaps this may be deserving of an apology, perhaps not. To me, however, it is the statement of fact and my answer to all criticisms and critics, and so far as I am concerned I believe my silence to be justified. I hope that from this out into the future, the criticisms will be based only upon experience on carefully conducted work with these methods, that the work will not become commercialized, and that this volume will encourage honest, hard-working and studious types of workers taking it up.

Considering the poor work that has been done on this subject in the past, and in the light of my experience and beliefs is still being done all about us today, I feel now that the time has arrived when record of this work should be presented. Inclusive as it may be in places, indefinite as it may be in parts of the subject, difficult as it is to describe in words a shifting and ever variable subject, I nevertheless have set myself to describe as best I can my own experience and beliefs on the subject. Time and time again, especially of late years, this life's work has presented limitations, barriers not possible to surmount and which seemed so easy years ago when the work was young and so much less was known about it by me. I hope that by this effort those who follow in the biologic aspect of this field of work (and it is worth a life's study) may now be more helped in their cases.

From a dubious start when all was doubt and question, the major part of the work seems finished, but one still constantly meets with troublesome matters here and there in which research is always essential, perhaps for understanding or additional data in just the single case, which, in even an ample experience in the work, may not duplicate itself again in one's whole life time of practice and study. My experience on the subject was that at the beginning there was much that was difficult of understanding and of the folly in the beliefs and practice as presented in the works of Bouchard, Combe and others. These being unscientific and in many instances stupid, I long ago discontinued them all and afterward handled the subject entirely in a biologic way. Along these lines it took years to obtain some sort of an understanding of it for even the simplest of starts. It was proven long ago that at no time is it a simple matter, like the result of an examination and a test for a diagnosis, a treatment, and a happy expectation of a cure or a worth-while result. Quite candidly also, at no time has it the essentials of a science, or the beginning and end of a diagnosis and a result in treatment. In place of these, experience, the only real teacher in it, must be depended upon for suggestions of the shifts to try for the results desired to be accomplished in either diagnosis or therapy. Ofttimes with the best of ef-

fort, the deepest of study, and the most painstaking and time-consuming of procedures, the laboratory cannot prove the clinical results that are accomplished, although most times these two would fit together.

I wish now to express my profound thanks to the many physicians who have referred cases to me these many years. Many times without their help, encouragement and assistance the results would have been nowhere near so happy. Some of these no doubt I have disappointed, and my only answer to them is the weak one of having done my best but failed, at times because the biologic diagnosis or therapy were wrong or the methods did not accomplish, and more times I think because failures were brought about because of misunderstandings or laxities on the part of these physicians and the patients. To the physicians of these patients, my thanks are nevertheless freely given. I wish also to thank the many technical workers, laboratory assistants, nurses and others who have helped along during these thirty years.

ANTHONY BASSLER.

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CHAPTER I.

GENERAL CONSIDERATIONS OF INTESTINAL TOXEMIA.

THE TERM "INTESTINAL TOXEMIA."

In beginning a discussion of this most important subject, let us first consider the terminology.

The reason the term "auto-intoxication," so familiar to lay people, is not employed in this volume is because its meaning is too broad. "Auto" signifies action of, from within, by or upon self, and in familiar usage "intoxication" pertains more particularly to states produced by the drinking of spiritous liquors, although in medicine it is used to represent constitutional poisonings such as septic processes. Unless one adds the term "intestinal," as did Combe, the term auto-intoxication could just as appropriately be employed for toxemias from apices of teeth, from the gall-bladder, kidneys, or any of the many organs in the body. "Chronic excessive intestinal toxemia," the term employed by Herter, is a far better one, but unnecessarily long and not apt in other ways. In the author's opinion, the best term to employ is simply "intestinal toxemia." This gives its source of origin and the term "toxic" means pertaining to or having the nature of poison. Of course, the term intestinal toxemia could be employed for tubercle bacillus, botulinus, anthrax and other straight types of bacterial infections in the intestines, but as the nomenclature of medicine has recently developed, more definite and specific terms are employed for these infections—thus the term intestinal toxemia can now be definitely employed to designate the states of disorder which the author desires to present.

BOUCHARD'S WORK.

The earliest writer in English on the subject and the one who first studied and took a deep interest in it was Charles Jacques Bouchard, as proven when he submitted the first

edition of his work in 1894. Collecting the data of many investigators, a work was presented which in a general way drew attention to toxic states. The major part of it had to do with the toxicity of urine, uremia, toxemias in typhoid fever, cholera, the causes of toxic fevers, and but little that is today worth while on the major subject of the intestinal toxemias. From his few lectures, however, that were devoted to toxemia of intestinal origin, he opened an interest in this subject in medicine, and while his work and beliefs are today no longer accepted, here and there are found gems of clinical thought and theories which will stand in medicine forever, and many of these are generally accepted as fundamental today. A worker in this subject, as well as many men in medicine, can still profit by a perusal of this old work.

COMBE'S WORK.

The work of this author was distinctly more specialized in character than that of Bouchard. To him belongs the credit of bringing out the thought of bacteria being of primary importance in the production of the aromatic bodies. His work is still quite complete in descriptions of various substances of toxic nature and the theories of the antitoxic functions of the body. He also brought out the suggestions of the defensive forces surrounding the intestinal canal, namely, the defense of the mucous membrane of the intestine, the liver, and what he designated as the "antitoxic glands." His work on the clinical side, as well as the examination of feces and urine, was much ahead of anything published before, and not a little since, even though in places he takes deep excursions into hypothesis. In treatment his dietetic suggestions are worth while, although the rules for fundamentalizing these are hazy and today not scientific. He was partial to medicaments to influence the vitality of proteolytic organisms, a part of the subject that will not stand criticism today. To him, as is well known, goes the credit of all that is known today about colonic irrigations (intestinal lavage), to which nothing new has been added by anyone. And he drew attention to the

value of physical agents and the lactic ferments in treatment. This brief array contains matters of first interest to a large part of the profession, and with them are many more or less important points still open to debate. The work should be read by the student of this subject.

SPA TREATMENTS.

Not much need be stated on this subject. For years literature has gone forth, mainly from the spas of France, Germany and Austria, on the efficacy of their waters in the treatment of intestinal conditions. The waters of many of these springs, whether drunk, introduced by rectum as simple enemas or colon irrigations, or by baths of various kinds, have been lauded as of benefit in intestinal toxemias. A careful study of the chemical composition of them, with full consideration paid to radium, iron, sulphate, arsenic and salt contents, leaves them hopelessly unattractive in this subject. For the hard-worked American business man and the woman who has worn herself out with social activities and "fussing," a sojourn at one of these delightful spas is generally beneficial to general health and in this way they may get themselves "on top" of an intestinal toxemia for the time being. Added to this would be the increased elimination effect on the bowel and kidneys from drinking waters, but so far as any definite or specific beneficial effect on a chronic intestinal toxemia is concerned, the less said about it the better.

COLONIC IRRIGATIONS.

The English translation of Combe's work on colonic irrigations appeared in 1910. For years before this time Combe employed the method in France, whence it quickly spread all over Europe. Over twenty years ago it was "the rage" among practitioners in Europe, and in every city and at every spa colonic irrigations were a popular method of treatment. The writings of Combe, the lack of knowledge on the subject of intestinal toxemia in general present at that time, and because there seemed to be nobody at this period who could analyze

the more important of the alluring and unwarranted statements of Combe, spread its popularity.

Before discussing the question of colonic irrigations in more detail, I desire to present Combe's full description, because by doing so it makes possible the answer to all the controversial points connected with it.

Quoting from Combe's volume, the following occurs :

Evacuation of the Proteolytic Microbes of the Intestine and Their Toxins by Intestinal Lavage.

The antiputrefactive regimen, and the critical or chronic intestinal disinfections, prevent in a very appreciable measure, the formation and accumulation of the products of nitrogenous putrefaction that appear in the intestine as a result of the action of the proteolytic anaërobic microbes.

But notwithstanding the unquestionable efficacy of these two means, they are not always sufficient to completely prevent the formation of intestinal poisons, and moreover they offer very little protection to the organism against the entrance of these poisons into the blood (intestinal auto-intoxication). The evacuation of the microbes of the colon and the toxins they secrete will fulfill this indication much better.

There are two ways of accomplishing this:

- (a) By lavage of the intestine.
- (b) By relieving the intestine.

(a) 1st. Lavage of the Intestine by Enteroclysis.

Enteroclysis or irrigation of the intestine is particularly indicated for obtaining this result.

For, as we have seen, nitrogenous putrefaction produced by the proteolytic microbes occurs only in the large intestine.

If by means of enteroclysis we succeed in properly washing out this portion of the intestine, we shall have the satisfaction of having fulfilled the indication presented.

Moreover, the water of the enteroclysis does not all pass out; more or less of it is retained in the intestine, sometimes as much as a half liter or more remaining behind.

This water will be absorbed and produce a lavage of the liver, kidneys and blood, as well as a rapid and abundant diuresis, which will carry off the waste products of nutrition and the toxins which had already penetrated the organism.

Lastly, enteroclysis serves as beverage and calms the thirst, inevitable during the first days of the regimen.

Therefore, in all cases of intestinal auto-intoxication, enteroclysis casts out *through intestinal lavage* the products of nitrogenous putrefaction existing in the colon, and through *diuresis* those that have already entered the general circulation.

This constitutes the first and principal indication for intestinal irrigation.

In the second place, enteroclysis is indicated in auto-intoxication due to muco-membranous enteritis, which is characterized by the formation of mucous shreds or membranes that are gorged with bacteria. These may also be the seat of considerable putrefaction, for they sometimes remain a long time in the intestinal folds before becoming detached and cast out with the stools.

A cleaning out of the intestine by an irrigation which would at the same time rid it of the mucus, and membranes with the microbes they contain, would consequently be most useful in relieving the condition. In the third place, intestinal auto-intoxication is accompanied in the great majority of cases by a spasmodic constipation, with accumulation of fecal matters which may be dry and occasion but little fermentation, but which may also exist, higher up, above the site of the spasms in the localized dilatations of the colon, in which the phenomena of putrefaction are extremely accentuated.

Here again the regular evacuation of these matters by enteroclysis will save the patient from complications, often serious.

Hence, the evacuation of the matters, the evacuation of the toxic products, the evacuation of the membranes, constitute the triple indication for enteroclysis. A fourth may be mentioned, but only for auto-intoxication due to chronic enteritis, this being an infectious inflammation of the intestine.

Enteroclysis, which can be made to carry the evacuating liquid up to the Bauhinian valve, may also be made to carry medicinal and microbicidal agents that are harmless to the patient.

That is the sole indication for the use of antiseptic enteroclysis.

Evacuating Enteroclysis.

Intestinal lavage is performed with normal salt solution (7 grams of salt to one liter of well-boiled water) at a temperature of 38° to 42° C., 100.4° to 107.6° F., according to the patient.

This solution is the one which irritates the intestine the least and which provokes the most abundant diuresis. For these reasons we prefer it to all others.

An infusion of flaxseed or marshmallow root may be added to it with advantage.

Technic of Enteroclysis.—To make a good lavage of the intestine, it is necessary to carry the liquid to the cecum, but without distending or irritating the intestine.

This cannot be done with the ordinary rectal douche, with short nozzle and strong pressure.

The large intestine, and particularly when diseased, is very contractile, and prolonged spasms occur in it from the slightest irritation.

Consequently, it is not surprising to see a violent irrigation under strong pressure produce spasms that not only prevent the tube from

passing, causing it to bend on itself, but even prevent the water from issuing.

We make use of English catheters of small caliber (16 to 24 French scale), but 50 centimeters long for children, and 1 meter or more for adults. They are pierced with an opening at the extremity and one on the side.

The catheter is connected by a short tube with a receptacle holding 2 liters, which is hung at the most 10 centimeters above the patient's buttocks. The patient should lie on the right side, with the legs flexed on the abdomen and the hips elevated.

The stopcock being opened, the catheter (properly lubricated) is introduced into the rectum for one or two inches, the flowing water opens the intestine beyond it and in a few moments more, it will be found easy to insert the catheter a little further and so on. By introducing the catheter slowly inch by inch, waiting each time until the water shall have opened the passage or straightened the bending of the catheter, its entire length can be easily introduced, and the water carried to the cecum without causing the patient the slightest pain or discomfort.

The quantity of water which should be introduced will vary greatly according to individuals and often in the same individual depending on the days; sometimes it may be 500 cubic centimeters, at others 1500 cubic centimeters.

Any back flow in the receptacle is a sure sign of intestinal spasm; if pain or discomfort of any kind should supervene during the irrigation, it should be stopped and postponed, even if the quantity of water injected is insufficient.

The irrigation should be voided by the patient at the first urgent inclination.

In most cases a good third remains in the intestine. It is well to precede the irrigation by a small evacuating clyster.

Indications for Enteroclysis.—We have seen that the indications for irrigation were the evacuation of the stools, membranes and toxic products.

Which means that all cases of auto-intoxication do not require intestinal lavage, and that enteroclysis in nowise forms part of its specific treatment.

We make use of irrigations in the three following conditions only:

1st. When there exists at the same time constipation and intestinal auto-intoxication characterized by high curves of the aromatic bodies; in these cases irrigations are quite indicated and they bring about a rapid improvement.

2d. Irrigations are indicated when there is a considerable degree of auto-intoxication, even though there are but few membranes and the stools are regular. Their use brings about a favorable modification in the general condition, and a disappearance of most of the discomforts.

3d. When enteritis is accompanied by many membranes and much mucus; irrigations are of great benefit, for they bring about a rapid sedation of the pains.

But when constipation is the only indication, irrigations of oil are much more active and present fewer disadvantages.

When the auto-intoxication is mild or benign, we do not advise irrigations, for the regimen and disinfections will suffice to bring about a cure.

As a matter of fact enteroclysis is not an indifferent remedy.

Badly done, it distends the intestine and increases its atony; if too great pressure is used it increases the spasms; repeated too often it irritates the bowel and exaggerates the mucous hypersecretion; too hot or too irritating, it provokes an artificial membranous enteritis.

These various reproaches are all well founded, but as Langenhagen wisely remarks, it is more due to the manner in which the lavage is done than to the procedure itself.

If enteroclysis is performed with the indicated precautions, most of the disadvantages disappear; but it is none the less true that they should not be abused.

It is necessary to know when to prescribe irrigations, at what intervals they should be made and to cease when the indication disappears, so as to disaccustom the intestine to their use, for too prolonged usage of the tube maintains the intestinal irritation.

Enteroclysis should, under those circumstances, be administered every two days only, then on the third day, etc., until it may be discontinued altogether.

Contraindications to Enteroclysis.—Irrigations are contraindicated in severe enteroptosis and in intestinal atony, when the water penetrates easily but is not voided.

When the intestine is irritable, lavage produces spasms of the colon with reflux of the injected fluid into the fountain or receptacle; the spasms are frequently painful and contraindicate the use of irrigations.

Lastly, they are contraindicated in certain impressionable patients with exaggerated intestinal reflexes in whom irrigations produce such severe pain that they are often accompanied by cold sweats, nausea and fainting.

Antiseptic Enteroclysis.

1st. *Irrigations of tannin* (1 gram to 5 grams per liter) may be used in the diarrheal forms and acute outbreaks. This substance, while not greatly antiseptic, is antitoxic; it precipitates the intestinal toxins into insoluble compounds and is slightly constipating. It is harmless, the only disadvantage of tannin being a tendency to slightly irritate the mucous membrane and cause intestinal spasms, often very harmful.

2d. *Irrigations with calumba* (10 grams per liter) offers the same advantages and presents the same inconveniences as tannin.

3d. *Irrigations with oxygenated water* (30 to 40 grams hydrogen peroxide per liter) are useful in acute outbreaks when the stools are extremely offensive.

4th. *Irrigations with ichthyol* (5 to 15 grams of a 1 per cent. solution per liter) give good results from the standpoint of intestinal disinfection, but they are often irritating to children.

5th. *Irrigations with borated water* (30 to 40 grams per liter) were studied and advised by Rovighi; they are useful but dangerous, several authors having observed phenomena of intoxication.

Résumé.

When the regimen and the disinfections are insufficient to control intestinal auto-intoxication, advise the use of:

- 1st. *Intestinal lavage with normal salt solution in the mild cases.*
- 2d. *Irrigations with ichthyol in severe cases.*

(b) 2d. Relieving the Intestine.

To control the constipation is for many patients and often for their physicians, the principal end to attain.

Constipation, it is true, is frequent in auto-intoxication, but as we have seen, it only plays a very obscure part in its pathogeny.

Nevertheless, it is necessary to combat constipation in all moderately severe intestinal putrefactions.

In the great majority of cases of intestinal auto-intoxication, the constipation is not of an atonic but of a spasmodic nature.

This fact is of the highest importance from the therapeutic standpoint.

Inefficiency of Enteroclysis.—Taking the above statements up in some detail I desire to present the following criticisms. The antiputrefactive regimen mentioned by Combe is but rarely beneficial, and when it is, it is only slightly and temporarily efficient. His two ways of accomplishing control of the proteolytic microbes, the evacuation of them and their toxins “by lavage of the intestine” and “by relieving the intestine,” are just about as efficient a cure as coughing and expectoration would be to benefit or cure pulmonary tuberculosis.

Had the last word been said on this by Combe, we would not have had all the justifiable debate on it that there has been, and which is most active today among even otherwise sound men of medicine, and which the future will inevitably show is more of a dream of desire than a fact of clinical reality. Enteroclysis or irrigation of the intestine, which, according to Combe and others, is so indicated in this microbe and toxic question, is in reality contraindicated in all intestinal toxemias. A little relief may come to some, but a cure never, and only too often more mischief is set up by these irrigations. The intestinal canal in anatomy, function, and clinically, was never made by nature for colonic irrigations, and no real disorder has ever been cured by them. If there is not much wrong

with a person they will probably be as beneficial as an enema. But, unlike an enema that costs a cent or two, the irrigation costs three or five dollars, is a much ado to accomplish, and requires an attendant to perform it.

Combe and others state and maintain that the proteolytic organisms occur in the large intestine. In practically all distinct forms of intestinal toxemia the main location of the toxemia is in the lower end of the small intestine which no irrigation by rectum would reach. The water of the enteroclysis irrigation that is retained, sometimes as much as half a liter, is supposed to "lavage the liver, kidneys and blood." That some of it goes into the body there is no doubt, and with it go a lot more toxic substances than would gain entrance into the system if no irrigation water were absorbed from the large bowel. Time and time again we have proven the body to be more toxic after an irrigation than when none is given. For an irrigation to be given "to allay thirst as a beverage" is a poor way to take a drink, and is a poor substitute for a hypodermoclysis, an intravenous or a "Murphy drip" in an extreme case.

Dangers in Colonic Irrigation.—The next indication for the colonic irrigation given is in the treatment of "mucous membranous colitis"—nowadays we more properly simply say colitis. Many practitioners believe this to be a valuable method of treatment for this condition, while the fact is that there is no better way to keep a colitis going than by it. Every colonic irrigation is followed by the discharge of mucus that is set up by its irritation. Witness the discharge of mucus in perfectly normal people following one. Not only is more mucus manufactured by the colonic irrigation, but why must it be brought away in the true case of colitis when mucus is a protective substance to save the intestinal tract?

Even if the mucus in a colitis is washed out, what good does it do? It is a good thing to talk about and impress the patient with, and in that way it may have a good mental effect, but no honest physician concerns himself much or allows himself to be influenced by these considerations. Even a nurse who does the work should be above such standards, and no

nursing school should stand for inculcating such commercialistic standards in the handling of ill people. Of course, ignorance of intestinal disorders on the part of physicians and nurses does allow for much, but is it not more honest to acknowledge this even with an ignorant patient?

It is stated that in spastic constipation good may come from these treatments. Careful research work has shown that spastic states in the bowel come from types of toxemias and other factors, largely in a neurasthenic type of individual. How can irrigations cure these, and how many hundreds of times have I seen added harm come from them?

About fifteen years ago when the fad of colonic irrigations in Europe had died out so that nobody, excepting an occasional bath attendant, was engaged in giving them, suddenly it was started all over again in this country by a few people who seized on a public made susceptible by Lane's work and his propaganda on the bowel "being a swill barrel." The people in America knew practically nothing about these treatments. They had read a lot about Lane's work (and he had some rather good broadcasters on it here that kept it up), and two or three men wrote medical articles on colonic irrigations, books appeared, charts were shown, and a warm controversy started in the profession as to whether the tip of a colon tube could pass to the head of the colon or not. In a short time special colon tubes were devised, convenient tables were invented, much was said of the efficacy of this or that solution, and the public seized upon these treatments as a "great find," "just what had been long hoped for," and a number of gastroenterologists changed from passing a stomach tube by mouth to that of twisting in a larger one by rectum to cure almost everything. A treatment popular long ago and obsolete in Europe suddenly was rediscovered in the United States, and an ignorant and always too gullible public lay down for it physically and financially like a heathen before his gods. Of course Combe got no credit for devising it originally.

New discoverers and administrators sprang up all over the land. Soon this or that colon irrigator began to be talked about, even in the profession, as a person who could do the

unusual this or that with a colon tube, and nobody seemed to know that Combe had done the same thing twenty years before him or her, and that none of the rediscoverers were accomplishing anything more. I would like that the reader here reread what is stated above in Combe's book, compare it with the recent medical literature on this subject in this country and prove it to himself. Note in his context that it "is not an indifferent remedy" and his remarks on "contraindications," and then compare them with how little is said on these in our literature, and how many cases of the contraindications are "cured or markedly benefited" by these attendants today.

When the conditions had reached such a pass that at not a few of the parties given in New York City (and no doubt elsewhere) in the winter of 1927-1928 the hosts announced to their guests that they should eat and drink all and whatever they desired, because at 11 o'clock colonic irrigators, one for men and one for women, would be ready to wash out those who desired it, it seemed to me that the time had come for the profession to learn something about this and discourage the use of such treatments.

In my thirty years of work on this subject I have not employed a colonic irrigation as a means of treatment of an intestinal toxemia, not even to give temporary relief. And in this time I have seen hundreds of people who have been so treated without benefit, and many made distinctly worse by them. Omitting from consideration the hundreds of cases of tuberculosis, cancer, pyogenic infections, ulcers and others, and also the many in whom colons had become irreparably crippled by the treatments, and the exhaustion, anemic and debilitated conditions caused by them, I wish here to state that their employment is a travesty on the science of medicine and a wholesale fallacy as a method to cure intestinal toxemias. To the physician who is keen about these treatments, whatever the temporary benefits have been, my advice is to learn something about the scientific side of this subject. To the nurses who make a living this way I say that they know not what they do, and they have been trained to do better sorts of work than this. To the "colon filling" stations where colons are washed

out for so much each time and a ticket punched that costs so much for so many of them, I would repeat that they are not only a menace to the profession, but particularly are they a menace to the public health. And to all I state that as the fad died in Europe so will it die here.

I have an abiding faith in the people. It takes a long time for them to learn about medical things. In this subject by a combination of enthusiastic and ignorant propagandists, costs of treatments, loss of time to have them done, their failures to accomplish anything worth while, and the advancing of scientific knowledge and better methods of handling these conditions on the part of serious physicians, the fad must die out here as it did abroad.

In addition to this, however, the medical profession has a duty to the public to discourage these treatments. To prescribe them or engage in giving them in any liberal sense is an evidence of ignorance of biologic intestinal conditions, and it would be best for the physician not to disclose such ignorance to the patient by recommending irrigations for him or her. This is especially proper advice to the specialist in stomach and intestinal diseases who is engaged actively in these treatments—and the country is full of them. Imagine a worth-while estimation of a nose and throat specialist today who employed irrigations or watery sprays to cure all nose and throat conditions, a urologist who washed out the urinary bladder of all patients for the cure of urinary tract conditions, or a gynecologist who employs vaginal douches to cure all gynecological conditions. These being manifest, what better can be said about the general practitioner, the internist or the gastroenterologist who employs colonic irrigations to treat intestinal states? If a medical man's knowledge of intestinal states does not go beyond this, one of two things is necessary: either he should begin a scientific study of these conditions so as to learn something about them, or he should discontinue handling these cases.

As with an enema, an occasional colonic irrigation can do no harm, but as a means of treating chronic intestinal states, far better let the medical man leave these treatments in the

hands of the commercialistic nurses, physical culture parlors, bath-gymnasiums, and the non-qualified side shows of medicine.

ACIDURIC BACTERIA INSTILLATIONS AND INJECTIONS.

Bulgarian Bacilli.—Just a few years ago, as a consequence of a belief expressed by Metchnikoff, the so-called *Bacillus bulgaricus* was vaunted as the solution of this problem. Pharmaceutical houses promptly got busy devising means to sell an advertised product under a proprietary name, the basis of which was the content of this organism. The various drug stores in large placards advertised this or that product of milk fermented with this organism, and in various scientific quarters studies were made to get the products and the organism perfect for human ingestion. In the hospitals the physicians—who as a rule are poor students of intestinal toxemias—were feeding patients on these milks and claimed wonderful results from their use. Patients came to the office with hip flasks of this milk on them just as toxic as before they had started with its use. Thousands of people were “B.B.” culturing their intestinal canals to cure intestinal conditions and to live long. As time went on millions of dollars were spent and nobody got much if any benefit, although many were hypnotized into the belief that something was being accomplished because so many physicians were recommending its use; there was such a broad interest in it; and so many were using it. Time, trial and experience finally brought this fad to its termination, and today one hears less and less about the use of *Bulgaricus bacillus* milk.

At the time the fad was at its height, I made some observations on stools of patients who had taken this milk; some in which the milk alone had been taken and others where the milk had been taken in abundance with foods. In a large number of observations the *Bulgaricus bacilli* could not be recovered from the stools, they having died shortly after their ingestion. As a result of these observations of mine becoming known by manufacturing concerns, the organisms were then added to petroleum jelly so that they would take on an

oily coat to keep them alive and carry them down, and on these products a recrudescence of the fad took place. This ran along for a while until two new ideas came along, the use of *Bacillus acidophilus* and mineral oil.

Bacillus Acidophilus.—As a result of the pioneer work of Moro and Tissier, it was proven that the infant in health harbored large quantities of *B. acidophilus* of Moro and the *B. bifidus* of Tissier in the intestinal canal. To this Mereschkowsky attached considerable importance, regarding them as being protective inhabitants of the digestive tract. It took about fifteen years for this assumption to take root and grow sufficiently into an attempt to secure definite results in the suppression of harmful organisms by their use. Knowing that many toxemias, especially those of the putrefactive forms, are alkaline conditions, this was enough to start the whole fermented milk affair of the B.B. culture days over again.

What particularly helped in this were the publications of Rettger and Cheplin, added to by several others, and at the time of this writing, *acidophilus bacilli*, various products of them and the milks fermented and containing them, occupy a height of popularity in the profession and the lay mind today as did *Bulgaricus bacilli* years ago.

However, this much can be said of *B. acidophilus*, which is not true of *B. bulgaricus*, that when it is ingested in large quantities about half of the intake of the bacteria will pass from the rectum in more or less viable form and that they do tend to keep the intestinal contents acid, thereby somewhat controlling the alkaline products of putrefactions during the time they are being ingested regularly. But the facts that do not seem to be known are, that they do not destroy the putrefactive organisms (only hold their end products somewhat in abeyance for the time being), and one needs but to stop the ingestion of these organisms for a few days to find all of the toxemia return; that they are normal denizens of the intestinal canal of man found in all its levels and that simply the ingestion of dextrose (milk sugar) will bring about a practical elaboration of them without the ingestion of the organisms

by mouth; and that in the aciduric types of toxemia their use is worse than useless and contraindicated.

This latter statement is of much clinical importance, and it is because the use of *acidophilus bacilli* is encouraged and engaged in so promiscuously without basing their employment on a careful chemical study of the gut, that unless this is done their use had best be altogether discontinued. On the whole, they are a most inefficient method of treating even the simplest forms of intestinal toxemia, and in a few years will no doubt be supplanted by some other method and thus go into the history of medicine as did the Bulgarian bacilli. The diagnosis and best treatments for intestinal toxemias are far from being as simple as believed, and because serious work on them is not being done by the profession, fad after fad will continue to come along and eventually go. As to the administration of these bacilli by rectum, this is even more ridiculous than their use by mouth.

MILK AS A USEFUL FOOD.—Apart from the slight aciduric benefit that comes from the *B. acidophilus* in some types of toxemia, no one seems to have given the diet of milk the credit in these treatments that it deserves. For many, many years in medicine, back almost to the Middle Ages, milk has been employed as a food for the sick, especially those having fever. Why did the older clinicians use it so extensively? They found by experience that milk could be taken far more easily than solid food, in that way some status of nutrition being kept up, but more particularly that fevered patients on milk diets did well in most illnesses. Of late years the reason for this has been found, and it is that people on a milk diet harbor fewer organisms in their intestinal canals than when any other foods are taken.

Considering intestinal toxemias as a bacteriological subject, milk is an ideal food for these conditions, but not to the extent of "milk cures" for everything, kept up as in the foolish standard of some of our sanitariums. But, as careful stool examinations have proven, toxic people on a milk diet will for the time being pass hardly a third of the dried weight of organisms in their stools compared to other foods, the reason

for this being that milk is so thoroughly digested, even in the upper levels of the intestinal canal, that there is much less residue for bacteria to live on and thus propagate in the lower levels. Added to this there is no cellulose detritus. Also important is the fact that milk contains lactose (milk sugar) and this stimulates the growth of the *B. acidophilus* as well as the *B. bifidus*, and these then would somewhat acidify an alkaline toxemia with benefit for the time being.

Why then give the *B. bulgaricus* or the *B. acidophilus* the credit for accomplishing something which is accomplished almost totally if not completely by the milk alone? Why hoodwink ourselves and the public when these facts concerning milk as such are so positively true? Against this simple, yet definite fact, the profession has been a party guilty of the useless spending of millions of dollars of the people's money on fermented milks. Of the many questionable things in fads, fancies and poor work in the handling of intestinal toxemia, this simple matter of *B. bulgaricus*, *B. acidophilus* versus plain milk stands out alone as proof conclusive that the time has come for better work in these disorders to be done by the profession.

PURGATIONS.

There seems to have been present for many years the idea that intestinal toxemias are simply and wholly a constipation affair. To a considerable extent this is true in some of the secondary forms of toxemia, but even here it has nowhere near the significance that is generally believed. Taking a complete evacuation of the sigmoid and rectum once a day as a standard of normalcy, one nevertheless commonly sees this in the most toxic type of person, the same being true of those with more bowel movements, even to diarrheic states. On the other hand there are those whose bowels do not move for days and no toxemia is present. Often one meets with a patient who does not feel so well if he be constipated or does not have a daily movement. Usually these people have an intestinal toxemia and generally it is of the primary type. But there are others who are not toxic and in whom the lack of

movements has only an anxiety mental effect—"an autosuggestive type of toxemia."

Alvarez was largely correct on this question, excepting his article pivoted on the wrong end of this subject. He wrote of bowel movements when he should have undertaken to describe toxemias in this connection. Evidently he is not interested in detailed experience in intestinal toxemias or else does not believe that they exist. But in this much I agree with him, that regular bowel movements are largely a fetish with the profession and the lay public. Constipation is only of significance in connection with its cause, and if an intestinal toxemia is present.

For these reasons the employment of purgative or laxative drug substances is as much in error in connection with intestinal toxemia as colonic irrigations. They have, however, this added damaging factor, that in intestinal toxemia of primary forms they usually render the patient more toxic by their use. One needs simply to examine the urines of these people after a purgation to note that this is true; to observe the woman who, when her colon is congested initial to menstruation and more actively absorbing, to see that she is more toxic from the bowel at this time; and to study the few persons who feel badly after bowel evacuations. All purgation and laxative remedies bring about their results by causing irritation and bowel congestion. In the primary toxemias this in turn increases the resorption of toxic substances, leaving people more toxic for the time being, and in no way relieving or curing the toxemia, and most often heightening the running course of the toxemia.

There may be a relief of symptoms for the time being but in the end the patient is worse off than before he took the drugs. This is true of the steady use of every substance for the purpose that has been employed in the *materia medica*s from the Egyptian days, *cascara sagrada* being the least harmful of all. The use of purgative and laxative medicaments to treat the primary forms of intestinal toxemia is a mild form of malpractice. Every effort should be made to accom-

plish satisfactory movements by diet, and this should be long persisted in before any resort is made to medicinal means.

Mineral Oil.—Of late years, especially since the advice of Lane, various forms of so-called mineral oils have been used to accomplish movements of the bowels. Not being a medicament in the strict sense it possesses an attractiveness for this reason. Of course, as time went on, the manufacturing houses added reasons for the use of mineral oil in preference to other substances, such as, being an oil, when taken before meals it limits gastric acidity, it lubricates the bowel, which in dry colitis is worth while, and so forth. The main reason of its popularity has been its extensive recommendation by surgeons after operations.

For a quarter of a century, because of anesthesia and the impunity of operative procedures and antisepsis and asepsis, surgery has been paramount to medicine in the estimation of lay people, and many surgeons believe this themselves. The future will prove that as we medical men advance there will be less and less surgical work done, and internal medicine will come back to its former paramount position and never retreat again. However, when a surgeon who has operated upon an individual makes a remedial recommendation it generally goes far in the estimation of the patient, those around him and even with a great many medical men. This and the supposed harmlessness of mineral oil and advertisements in the lay press are the reasons for its popularity. It is, however, a worth-while remedy, although it does not cure constipation, does not absorb toxic products (which it is advertised to accomplish), and has no particular advantages clinically over medications of the simpler types. If kept up long enough it often diminishes nutrition and may even bring on states of anemia and debility.

Then again, the oil if taken in fair-sized doses often passes out of the anus leaving constipated stool in the rectum. On the whole it is a poor substitute for the dietetic handling of constipation, and in good medical handling its use is many times contraindicated. As a treatment for intestinal toxemia of the

primary type it has no value; for the secondary forms it may serve in a limited medicinal sense, but most times is valueless.

Yeast.—The newest fad now, kept up by extensive advertising in Sunday newspapers, is the ingestion of yeast. It is being vaunted as a cure for constipation, intestinal toxemia, debility, "that tired feeling," etc. Graphic pictures of intestinal canals, beautiful women, hospitals and physicians, and testimonials, etc., are all presented as are cakes of yeast on the cashier's desks in public restaurants. So far as the commercial and business side of this is concerned I have no brief, but so far as the advertisements of the profession are concerned, and the gullibility of the public, we have two excellent examples of the absence of scientific knowledge on this subject.

There are some individuals in whom an ordinary yeast infection of the intestinal canal will stimulate the bowel to more frequent movements, just the same as can be accomplished or is seen in the higher grades of yeast fungi infection. Sometimes with ordinary yeast a secondary toxemia is symptomatically controlled for the time being, a primary toxemia never. In the many infections of straight and mixed types I have studied in people whose intestinal canals are laden with yeast fungi, the patients not only continue to harbor the infections but commonly are made definitely worse. So far as vitamins are concerned, there are better ways through food and other substances to accomplish an intake, and I care not whether it is baker's or brewer's yeast that is considered. As long as the extensive advertising of this keeps up, the yeast fad will continue. When this ceases the fad will die out quickly, because it has practically nothing scientific or biologic to sustain it in this connection.

SUBCUTANEOUS VACCINES.

In 1910 I drew attention to the value of rectal instillations of living *B. coli communis* to benefit intestinal toxemias. This was based upon the theory expressed by Herter on work done under his direction, that the presence in the colon of immense numbers of obligate microorganisms of the *B. coli* type was an important defense of the body in the sense that they

hinder the development of putrefactive decomposition. This promptly led to a recommendation of their use by Turck for subcutaneous administration. This was taken up by the late J. Reece Satterlee and others. Manifestly, the only position these men took or persisted with was one opposed to both Herter and myself, namely that the *B. coli* were inimical instead of beneficial organisms to have in the intestinal canal. If not this, then why their use in a vaccine, by subcutaneous method on the Wright basis of immunity, opsonins, etc.? Of course, as the years wore on, more and more use was made of this vaccine method of treatment, more and more optimistic articles appeared, until by virtue of its inefficiency the method ran on the rocks and broke up in interest. It needed but little knowledge of the biology of this subject and intestinal activities, together with some fundamental knowledge of vaccine immunities, to realize promptly that it was doing something that started nowhere, led nowhere, and finished nowhere.

But other men elaborated on this erroneous premise of Turck and Satterlee, and carried it further. They developed various other vaccines, mostly combinations of colon bacilli, streptococci, pneumococci and staphylococci, and various biological concerns and commercial vaccine-making laboratories spread the interest in them still further. For the last several years the use of these stock vaccines in various combinations have been extensive throughout the world to cure most anything that may exist in the human intestine. This today is not so common in the United States as it was or as it now is abroad, mainly in the Latin countries. It is interesting to have patients report a certain stock vaccine that was used with benefit in a few doses, and then to have the experience of continuing with the same product with no more effect than injections of plain water. There are several excellent reasons for this failure, apparently not known to the users or the profession in general. Before entering into them I desire to bring out another vaccine development of very recent times which just now has the vogue and my answer to this will

be my answer to the failures of the above-mentioned methods also.

As a result of some worth-while biologic studies on chronic disease of joints and focal infections about the body, mostly in the head, Burbank and Hadjopoulos advanced serological diagnosis methods and vaccine therapy in the treatment of osteitis deformans; etc. While they properly cultured stools as well as discharges from the tissues of the head, throat, etc., for study, diagnosis, and therapy, their work was always in connection with the arthritides. To me Burbank's work is logical and his studies possess the value of being confined to an entity of disorders (joints). At no time, although he recognized that the intestinal canal could be an important source of focal infection and toxemia, did he suggest or carry his work into definitely undertaking to cure an infection of the gut by his methods of diagnosis or treatment. Evidently Burbank recognized the fact or accumulated the experience that so far as the intestines were concerned no benefit could come or be depended upon by these vaccine treatments. But this was not so with others. Promptly these entered the intestinal field to utilize the methods Burbank employed in the arthritides, to diagnose and cure intestinal toxemias. In such, in rapid stages, an interest and employment has spread, and it is the popular method of today—another one that will die away like the many others.

I desire first to draw attention to a typical diagnostic chart of a patient from one of the laboratories, generally sent to men who have charge of a patient.

Complement activity. 3 plus.

Antibacterial unit. 4 plus.

Stock antigens:

1. *Aërobacter aërogenes*. Negative.
2. *Aërobacter aërogenes*. Negative.
3. *Aërobacter aërogenes*. Negative.
4. *Aërobacter aërogenes*. Negative.
5. *Aërobacter aërogenes*. 2 plus.
6. *Aërobacter aërogenes*. Negative.
7. Typical non-hemolytic coli. 4 plus.
8. Typical non-hemolytic coli. 4 plus.
9. Typical non-hemolytic coli. 3 plus.
10. Typical non-hemolytic coli. Nega.
11. Typical non-hemolytic coli. Nega.

12. Typical non-hemolytic coli. 3 plus.
13. Typical non-hemolytic coli. Nega.
14. Typical non-hemolytic coli. 4 plus.
15. Atypical coli. Negative.
16. Atypical coli. Negative.
17. Atypical coli. Negative.
18. Atypical coli. Negative.
19. Atypical coli. Negative.
20. Atypical coli. 4 plus.
21. *Esch. acidi-lactici*. Negative.
22. *Esch. acidi-lactici*. 3 plus.

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| 23. Narrow hemolytic coli. 4 plus. | 63. Non-hemolytic enterococci. 4 plus. |
| 24. Narrow hemolytic coli. 2 plus. | 64. Non-hemolytic enterococci. 4 plus. |
| 25. Narrow hemolytic coli. Negative. | 65. Non-hemolytic enterococci. 4 plus. |
| 26. Narrow hemolytic coli. Negative. | 66. Non-hemolytic enterococci. 4 plus. |
| 27. Narrow hemolytic coli. Negative. | 67. Non-hemolytic enterococci. 3 plus. |
| 28. Narrow hemolytic coli. Negative. | |
| 29. Wide hemolytic coli. Negative. | 68. Hemolytic enterococci. 4 plus. |
| 30. Wide hemolytic coli. Negative. | 69. Hemolytic enterococci. 4 plus. |
| 31. Wide hemolytic coli. 4 plus. | 70. Hemolytic enterococci. 4 plus. |
| 32. Wide hemolytic coli. Negative. | 71. Hemolytic enterococci. Negative. |
| 33. Wide hemolytic coli. Negative. | 72. Hemolytic enterococci. 4 plus. |
| 34. Wide hemolytic coli. Negative. | 73. Hemolytic enterococci. 4 plus. |
| 35. Wide hemolytic coli. Negative. | |
| 36. Salmonella. 1 plus. | 74. C. Welchii. |
| 37. Salmonella. Negative. | 75. C. Welchii. |
| 38. Salmonella. 2 plus. | 76. C. Welchii. Type III. Negative. |
| 39. Salmonella. Negative. | 77. C. Welchii. Type IV. 1 plus. |
| | 78. C. Welchii. Type IV. 4 plus. |
| 40. Eberthella. Negative. | 79. C. Welchii. Type IV. 4 plus. |
| 41. Eberthella. 3 plus. | 80. C. Welchii. Type IV. 4 plus. |
| 42. Eberthella. 4 plus. | 81. C. Welchii. Type IV. 3 plus. |
| 43. Eberthella. Negative. | |
| 44. B. typhosus. 1 plus. | 82. C. sporogenes. |
| | 83. C. sporogenes. |
| 45. B. paratyphosus B. Negative. | 84. C. histolyticum. |
| | 85. C. multifementans. |
| 46. Hemolytic streptococci. Negative. | 86. C. bifermentans. |
| 47. Hemolytic streptococci. 4 plus. | 87. C. centrosporogenes. |
| 48. Hemolytic streptococci. 4 plus. | 88. C. putrificum. |
| 49. Hemolytic streptococci. 4 plus. | 89. C. novyi. |
| | 90. C. tertium. |
| 50. Non-hemolytic streptococci. 4 plus. | 91. Staphylococcus albus. 4 plus. |
| 51. Non-hemolytic streptococci. 4 plus. | 92. Staphylococcus albus. 4 plus. |
| 52. Non-hemolytic streptococci. 2 plus. | |
| 53. Non-hemolytic streptococci. 4 plus. | 93. Staphylococcus aureus. 4 plus. |
| 54. Non-hemolytic streptococci. 4 plus. | 94. Staphylococcus aureus. 4 plus. |
| 55. Non-hemolytic streptococci. Nega. | 95. Staphylococcus aureus. 1 plus. |
| 56. Non-hemolytic streptococci. 4 plus. | 96. Staphylococcus aureus. Negative. |
| 57. Non-hemolytic streptococci. 2 plus. | |
| 58. Non-hemolytic streptococci. 4 plus. | 97. B. Friedlander. 4 plus. |
| 59. Non-hemolytic streptococci. Nega. | |
| 60. Non-hemolytic enterococci. 4 plus. | 98. B. pyocyaneus. 3 plus. |
| 61. Non-hemolytic enterococci. 4 plus. | 99. B. pyocyaneus. 4 plus. |
| 62. Non-hemolytic enterococci. 3 plus. | |
| | 100. Alcaligenes. 4 plus. |
| | 101. Alcaligenes. 4 plus. |
| | 102. Proteus. 4 plus. |
| | 103. Proteus. 4 plus. |

Patient's own antigens:

- | | |
|----------------------------|--------------------------------|
| 1. B. coli. | 6. Wide hemolytic coli. |
| 2. Aërobacter aërogenes. | 7. Non-hemolytic streptococci. |
| 3. Atypical coli. | 8. Hemolytic enterococci. |
| 4. Non-lactose fermenters. | 9. Non-hemolytic enterococci. |
| 5. Narrow hemolytic coli. | 10. C. Welchii. |

At first glance the above looks like a worth-while bit of work and to the uninitiated it generally passes as such. Let us see. Practically everyone of the bacteria mentioned are or can be denizens of the intestinal canal against which complement-fixation tests may be positive. This being true, of what value is a positive or negative result? On the other hand, one commonly sees straight infections of the non- and hemolytic coli, the non- and hemolytic streptococci, the Welchii and staphylococci, even with marked ulceration and toxemia, with perfectly negative fixation tests. Why? Because the anti-toxic and defensive factors within the bowel, in the mucous membrane, liver, and general body neutralize the toxic products from them.

With this are the immensely important symbioses of bacteria in the intestine which are definitely misleading in complement-fixation diagnosis. An organism, ordinarily of significance, may be perfectly harmless and innocent in the intestine and the complement fixation be positive. This same organism may symbiose with another or others, perhaps innocent and perhaps not, perhaps of the significant infecting types and perhaps not, and a true toxemia still exist. What is interesting now is that the complement-fixation tests may be negative as to all in this combination because the toxic substance then produced is not specific to any of the organisms that entered into the toxemia; and yet it may be, and since there is no way of telling, I fail to see how the fixation tests can be of any total or depended-upon value in a clinical sense.

If any test is found so often negative in a positive case, and positive in a negative case, as these complement-fixation tests prove to be, their use for the purpose should be discontinued. This irregularity enters into all of diagnostic fixation work no matter where the infection is situated or what its character may be. But in infections elsewhere in the body than the intestine, a fair degree of regularity exists. When, however, the infection is in the intestine there are so many modifying processes operating on the toxic products, that the tests are no longer specific as to bodies in the blood serum, and one deals with such a bacterial maze in the intestine that defini-

tion is clinically not worth the effort. Nobody recognizes this fact concerning the intestines more definitely than Burbank himself, and although he is quite frank about its lack of value, the profession is still misleading itself in the error of it concerning the intestines.

Final factors of importance here are that many of the organisms that are potential infecting agents in the bowel are endogenous and thus not capable of producing toxic substances, at least not from within themselves. Therefore, what is the value of complement-fixation work when there is nothing in the blood to figure them by or combinations or body-resisting substances that you cannot get tests on? Finally, almost all intestinal bacteria are influenced by character and quantity of foods for a short time. Thus the foods an individual may have eaten the day before the blood was taken may give a positive fixation reaction, and the day after be normal. And added to this are the irregularities of most all intestinal bacteria being facultative, the rate of transit through the alimentary tract as well as the chemistry of the foods that had been taken, the time of the day the blood is taken, and the unknown factor of the various strains and associations.

It certainly is the fact (and against this there is no debate) that the use of the complement-fixation methods of diagnosis and the employment of suggested vaccines, and the use of the still more empirical types of vaccine therapy, are about as valuable as a single thermometer observation of a higher than normal temperature is diagnostic of malaria, typhoid or rat-bite fever and the use of acetyl-salicylic acid for their cure.

DIETETIC FADS.

There is no one thing that has been more popular than different types of dietetic cures for intestinal toxemias. Institutions undertaking to accomplish this are spread all over the world and hardly any two of them are alike in set plan. At one place they depend mostly upon milk, sometimes totally; at another no meat, fish or fowl are allowed to be eaten; at

another the old Salisbury diet of beefsteak and water is depended upon; at another it is a question of roughage mixed with the food taken; at another it is entirely a matter of caloric feeding, and so on. So much has already been written on this subject of faddistic dietetic treatments and so little is worth the while, that a review of this literature would be valueless and would only serve to further confuse the subject.

To hold to an eliminative plan of diet in whatever way this would be fundamentalized is proof conclusive that poor quality of work is being done on this subject in that place or by this physician. In the ignorance on this line of work in medicine, no matter what the advertising or commercial reasons may be, one should not generalize on diets for these cases. There are cases markedly benefited by a high protein diet, others a high carbohydrate, others a hydrocarbon, others a caloric plan, and so forth. These should be according to the intestinal infection and chemistry that are present, the designation being according to whether the toxemia is putrefactive, fermentative, or the mixed type, and then one should not depend upon the diet alone or continuously. Significant points on these will be presented later.

In this connection I desire to bring out my belief that dietetic faddists and fad institutions may by their plans hit the right sort of procedure for cases here and there, but on the whole they fall short of a scientific chemical and biological handling of the subject. It is fortunate for many of them that the general run of their patients know nothing about this subject. To a patient a diet is a diet and it must be correct for the condition that exists, and it is fortunate for these faddists that there is this total lack of knowledge on the part of the public.

Patients have come to me after having been in three or four different well-known institutions, having been carefully examined in each, with careful stool and urine analyses, and having been dieted by each in opposite ways. One is led to the deduction that but little is gained from their examinations of stools in the way of dietetic suggestions, and, although a diag-

nosis of toxemia always seems to be made by them, that this is sufficient for the maintenance of the diet fads characterized in the place and not uncommonly maintained for all. This very much resembles the use of one classification of drugs in the materia medica being used for all diseases and conditions.

Definite Food Substances for the Treatment of Intestinal Toxemia.—What has been stated of faddist types of diets also holds good here. The vast majority of these are based on laxative effect and when effective in this way are generally of symptomatic benefit. In this class one finds a long list of foodstuffs which have been prepared in certain ways, the taste changed by cooking or roasting preparation or by additions of other substances. Generally they are put up in cardboard boxes or cans with most lurid labels, high-sounding names, and optimistic statements of their value. Very often a printed pamphlet accompanies them extolling their virtues still further, and, should you forget about it, the daily newspaper refreshes your mind. Some of these products have a value in certain ways, rarely, however, equal to the statements employed in advertising and commercialistic propaganda. Outside of those containing bran, lactose, the meat substitutes, or the other laxative ones, the less said about them in scientific ways and as curative toxemia products the better; and for those which contain substances of some symptomatic value one pays a high price for what could be purchased as "open stock" in grocery stores with as good an effect.

If increased cost is not a consideration, and one is not led astray by the advertised exploitation of their virtues, there are among them several quite worth-while patented articles which, as additions to diets from time to time, serve to good purpose in the general handling of this subject. Those containing lactose have a stimulating effect on the aciduric bacteria in the intestines, these being of benefit or of negative or detrimental effect according to the type of toxemia chemistry that is present. Those containing bran, agar, and psyllium seeds are laxative in most instances and usually more pleasant than bran as such. So far as meat substitutes are concerned, the curtailment of the amount of the native pro-

tein foods ingested answers all purposes. The best substitute for meat is less meat or none at all. This is cheaper than substitutes and better. Finally, no place or person can cure intestinal toxemias, especially of the primary types, by diets alone. It just cannot be done.

The Sansum suggestions of handling chronic bowel conditions pays no attention to biological errors or infections, and only the dynamic and dietetic sides of the problem. In them, roughage for bulk requirements, types of diets for so-called "acid-ash type of acidosis," the acetone type, so-called normal diets, and caloric, mineral, vitamin and water-requirement plans are stressed as cure-alls. By their employment the results are apparently successful and generally scientifically useless. The whole scheme of them is an array of shortcomings of the handling of chronic intestinal toxemias. The most important factors in proper diagnoses and treatment are those of infection and immunity production. In the last few years individuals are multiplying who have been on the Sansum plans of treatment with the infections continuing, and I anticipate, as time and propaganda continues, they will increase in numbers as these new ideas broaden. Since no attempts are made for biologic and chemical diagnoses, and the treatments are based totally on bowel dynamics and vague and almost valueless therapeutics, the cases of infection will continue. It may be well to repeat to those who follow such types of methods that intestinal toxemias are bacterial affairs and dietetic to only a limited degree and then but for a short space of time. No one can clean up a chronic infection by causing more frequent movements of the bowels or the reaction chemistry of the urine through diet; these results are not as often accomplished as Sansum suggests. In my management of intestinal toxemia by the Sansum methods and after an ample experience with these diets, their employment has been discontinued, the old plans of diets being far more useful, insofar as diets can help.

MENTAL AND PHYSICAL REST TREATMENTS.

While certain physicians and institutions who employ these methods are not advertising the facts, there are benefits of a temporary nature accruing from these types of treatment. A calm, tranquil life in the country, free from emotions and from occupations and cares of business, household or active social life are generally beneficial to these people. The usual so-called "rest cures" or the "rest and food cures" carried out by a number of our gastroenterologists today for anything digestive in character, generally conducted in some private sanitarium, answer quite well for a symptomatic relief of such gastric and intestinal symptoms as are due to vital and physical fatigue.

One must remember that so far as a toxemia is concerned these treatments would only be of benefit in temporary ways, and, in the majority of cases, an up-and-around treatment, especially when a fresh air and an exercise program is conducted, the patients do still better when biologically handled instead. In the primary intestinal toxemias the "rest" or "rest and food" cures are not only a waste of time and money, but distinctly an admission of limited knowledge of the subject, poor treatment and handling. It is unfortunate that so many of our more social types of internists and gastroenterologists have found it commercially worth while to "rest" or "rest-food" cure so many of our urban people. There is no doubt that with patients on low diets, those requiring dietetic control, and the fatigued ones, such types of treatment are occasionally worth the while, and there is no doubt that in many of our cases of intestinal toxemia fatigue is a common resulting state, and may need special attention. But to fundamentalize the handling of definite intestinal toxemias on these types of treatment is distinctly short-sighted medicine if engaged in seriously, because in the primary types of intestinal toxemia, no benefit comes that is substantial; in many of the secondary forms surgery is indicated, while the ambulatory types of medical treatment are distinctly more scientific and more worth while in the primary cases.

More pertaining to these treatments in intestinal toxemias when indicated will be presented later. In my remarks here I wish only to present criticism of the work of some men who have gone astray since the days of the late Jessie Meyer, and "rest" and "rest-food" everybody just because they have digestive symptoms and who do this also for the handling of intestinal toxemias. In intestinal toxemias, as a rule, the better the diagnosis, and the more specific and indicated the types of treatment, the less and less are "rest" and "rest-food" types of treatments required.

ENDOCRINE AND MEDICAL CONSTRUCTIVE TREATMENTS.

There are not a few men in medicine who treat intestinal toxemias by endocrines. Usually this is not done fundamentally on the toxemias, but, because certain gland imbalances exist, glandular substances are fed. In all of this work it must be remembered that in intestinal toxemias glandular deficiencies ensue secondarily and when you treat by these gland substances you simply feed a deficiency, generally obtain a poor result, and in no sense handle the primary causative condition. The same is true of tonics, "buck-up diets," and tissue constructive substances. Since all medicine is now agreed to employ diphtheria antitoxin in diphtheria infections, to solely employ a gargle or to prescribe the old iron and potassium chlorate mixture is looked upon as deficient medicine and poor handling. The same is true of employment of the endocrines and tonic and constructive methods in the curative handling of intestinal toxemias in general. More is presented on these matters later in this volume.

MEDICATIONS.

For a good many years the profession has attempted by different medicaments to bring about an "intestinal antisepsis," even though the germicidal effect of drugs in the intestinal canal is an ostrich proposition. The substances used for this purpose have been many just because none could be found that had any definite benefit. It must be manifest that in tracts like

the intestine, which is over twenty feet long, and one in which a number of phenomena take place, the hope of accomplishing "intestinal antiseptis" by any single or combination medications is doomed to failure.

Bouchard, more than any other, is to be regarded as the pioneer in this attempt, and he believed by charcoal, naphthalene, and iodoform taken internally it was possible to influence the proteolytic bacteria favorably to the patient. Baumann, who first used the method for determining ethereal sulphates in the urine as the gauge of absorption of putrefactive substances, was probably more responsible than anyone else for putting calomel so high in the list of the so-called "intestinal antiseptics," a place it has no right to hold based on any distinct antiseptic action.

A great array of drugs have been tested culturally, and reports pro and con have been expressed by many authors. For instance, Sucksdorff claimed to have obtained a drop of one-third in number of colonies in his own stool after taking 3.6 grams of quinine in divided doses; and even a more marked drop followed the administration of 2.1 grams naphthalene. Kumawaga, using 14 grams acetanilide, U. S. P., on a dog, reported a reduction of colonies in the proportion of thirty-seven to one. Salkowski got a considerable reduction of bacteria in the feces of a dog fed at intervals with chloroform water. Stern found no effect following naphthalene, betanaphthol, salol, and camphor. Von Mieczkowski, in cases of ileocecal fistula, tested the antiseptic values of bismuth, silver citrate, tannopin, betanaphthol, and menthol by mouth. With the exception of menthol, his results were that they were of no value; in the case of the latter there appeared to be a substantial reduction following the giving of such amounts as from 10 to 13 grams in divided doses.

As early as 1911, Assmann reported negatively on guaiacol cinnamate (styrakol), tannin-formaldehyde (tannoform), tribromphenol-bismuth (xeroform), and bismuth subgallate, using the culture method to gauge results. He employed dogs fed on milk-rice and meat-rice diets. In the same manner he tried the effects of two different silver-protein solutions (so-

called "colloidal" silver solutions), which had been superficially tested by Klimmer in 1900. If given in a solution of gum acacia along with the food, the results proved encouraging, as the colonies per milligram of feces fell at times quite low. In the giving of colloidal silver preparations to man, it ought to be borne in mind that absorbed silver might ultimately in part be laid down in the skin. In the dog, Klimmer states that absorbed silver is laid down in the intestines, liver, and spleen, but not in the nervous system, muscles, ligaments, or skin; in the dog the skin is devoid of sweat-glands, and this may be sufficient explanation why no silver is deposited there.

By the Strassburg technic the following agents have been literally "weighed in the balance and found wanting": naphthalene, thymol, silver citrate, betanaphthol, and salicylic acid (doubtful); magnesium dioxide and iodo-anisol by Feigen; in cases of intestinal catarrh, negative results were obtained by Friedenwald and Leitz using salicylate of bismuth, salol, potassium guaiacol sulphonate (thiocol), acetyl-salicylic acid (aspirin), and thymol; while favorable results with bichloride of mercury and ichthyol albuminate (ichthalbin) were noted; but the sharpest fall in bacterial excretion was obtained in a case of intestinal catarrh with hyperacidity when the patient was simply put on a change of diet. Berger and Tsuchiya reported a lessened bacterial output by the use of patented preparation of agar-agar and hydrogen dioxide. Steele found that in normal persons the administration of betanaphthol and bismuth salicylate caused a slight fall in the bacteria excreted.

Manifestly, it is most unwise to expect to change the status of affairs in the intestinal canal by means of any medicinal substances by mouth. This entire method and line of endeavor should be given up as unscientific and impractical. Such benefits as are brought about by use of the so-called intestinal antiseptics, singly or in combination, are due entirely to the effect upon the patient's mind, and not to biochemical change that these drugs bring about.

One deals with so many factors—the types of food, their physical as well as chemical characteristics, the age of the in-

dividual, the general condition of the body at the time, the physical expenditures of the muscular system which correct small digestive errors by balancing nutrition of the tissues, the various phenomena of digestion, the transit through the gastrointestinal tube, the chemical changes that are brought about by the various secretions of the canal, the incorporation of the bacteria with food, perhaps mucus, and the segmentation of the food in various sections of the canal, which a single dose or even small incorporated doses would not affect—that to continue the giving of intestinal antiseptics should be discouraged.

There is a great difference in the proportion of a disinfecting agent required to destroy microorganisms and that needed to restrain development. For example, the germicidal strength of creosote is about 1 to 300, but it is distinctly inimical to bacterial growth when present in the proportions of 1 part to 4000. The following table gives the generally accepted strength in which some of the intestinal antiseptics are positively efficient, and the dose this would represent for a volume of 6000 cubic centimeters:

	Antiseptic strength.	Dose required.
Betanaphthol	1 to 10,000	9 grains
Copper sulphate	1 to 1100	80 grains
Chlorine water (U. S. P.)	1 to 16	12 fluidounces
Creosote	1 to 3000	30 minims
Phenol	1 to 700	3 drams
Salicylic acid	1 to 1000	90 grains
Phenyl salicylate	1 to 800	115 grains*
Solution of formaldehyde (U. S. P.)	1 to 2800	31 minims
Resorcinol	1 to 2000	45 grains
Thymol	1 to 1500	60 grains

*This figure is based on calculations from the amounts of phenol and salicylic acid in this compound. Bouchard found by actual experiment that it required 75 grains.

These figures are based on the supposition that all of the drug administered will remain as such in the intestinal tract, which is, of course, contrary to the fact. The questions of the rapidity of chemical change and absorption become of much importance in judging the efficacy of this group of remedies. Is there evidence that any of the so-called intestinal antiseptics

can remain in the alimentary canal long enough to exercise their antibacterial influence? There is abundant proof that many of these substances are absorbed, in part at least, in a comparatively short time, but the evidence that any of them tarry for a considerable period in the bowel is far from satisfactory. The most important work on this subject that I know of is that which is offered by the ingenious experiments of Mieczkowski. From the presumptive evidence deduced from this it would seem that the conclusion is justified that theoretically it is possible to exert a mild degree of antiseptis in the intestinal tract, although it is unreasonable to expect any distinct germicidal influence. Clinically, though, this is not borne out to any practical degree.

In the experiments which Sucksdorff conducted, an effort was made to influence the number of bacteria in the alimentary canal by plating a weighed sample of the stool and counting the number of colonies in the usual manner of estimating bacteria and administering one of the following in the table that he gives:

WEIGHT OF BACTERIA.

Case.	Without drug.	With drug.	Drug.
1.	7.44 grams.	3.26 grams.	Bismuth salicylate.
2.	5.0 grams.	1.15 grams.	Bismuth salicylate.
3.	2.74 grams.	1.17 grams.	Bismuth salicylate.
4.	2.23 grams.	0.90 grams.	Betanaphthol.
5.	2.51 grams.	1.44 grams.	Betanaphthol.
6.	1.69 grams.	3.51 grams.	Betanaphthol.

From the above, three substances turned out as being apparently efficient in doses that are within the limits of safety. These are betanaphthol, formaldehyde, and creosote. It is probable, however, that formaldehyde is taken up with great rapidity, so that a practical intestinal antiseptic action would not be accomplished. Creosote, on the contrary, is absorbed with a fair degree of rapidity. It is possible by enteric coating to delay the absorption of this from the intestinal canal.

Betanaphthol is rather an insoluble substance, probably somewhat more soluble in the content of the bowel than in water. It is evident, however, that a substance which is so

sparingly soluble as this, must go into solution in the intestines very slowly and therefore linger in the bowel for a considerable length of time. For this reason, as well as the fact that even in very dilute solution it exercises an antiseptic influence, it would seem to be identified as the remedy of choice in cases in which we wish to influence bacteria in any part of the intestinal tract below the upper duodenum. Sternberg found that a 1:2000 solution destroyed the vitality of the cholera spirillum in 5 minutes, a 1:3000 in two hours, and a 1:16000 solution prevented the germination which a 1:24000 solution failed to do. These concentrations would correspond to doses of about 9 grains, 5 grains, and 3 grains, respectively. Briefly, the use of medicaments termed "intestinal antiseptics" and any efforts to find such a substance or accomplish such results medicinally should be discontinued. There never was, is not today, and never will be found any medicinal substance that could accomplish it, even in a small way.

CHAPTER II.

INTESTINAL TOXEMIA—CRITICISMS AND ARGUMENTS.

CRITICS—SOME POINTS ON WORTH-WHILE WORKERS IN THE SUBJECT.

Perhaps the most severe opponents of the intestinal toxemia theory are the laboratory pathologists. Not usually conversant or experienced in clinical medicine, nor brought into intimate contact with medicine through their services in laboratory procedures, nevertheless as a class they comprise the most severe critics existent, and by virtue of their position, scientific training, and work, many medical men listen to their arguments and often take them as granted facts. Simmering down all of the arguments and statements I have heard, their main contention is based on the fact that no toxic substance has ever been isolated to prove the existence of these clinical conditions.

In superficial thought this seems to be logical enough, but it will stand no depth of analysis. Anatomy, physiology, histology, pathology, and bacteriology are fundamental departments of medicine. Roentgenology in the hands of capable men is practically established on the same basis. But the department of medicine up to today that has rendered the least practical service to clinical medicine is chemistry, and it is generally to the chemist type of pathologist that such critics belong. They largely have drawn upon Koch's laws, which, while a proper basis for many biological matters in medicine, is not a proper basis for consideration of chemistry as an applied science to medicine. Medicine is largely a biological affair and the body is capable of so many protective forces, not at all estimable or provable by chemistry, that chemistry as an applied science to medicine has to go a long way in development before it will be helpful in diagnosing or estimating disease. At the same time it can be proven that there really

is no portion of medicine that chemistry has had more to its credit than in the estimation of intestinal toxemia.

To mention just a few, one has the estimation of the ethereal bodies, oxalates, the reactions of urine, and the tests and reactions showing the presence of the large number of various toxic chemical substances in stools which are abnormal in kind or quantity. To these must be added here and there of significance, different constituents of blood, in the hemoglobin, uric acid, red blood cells, etc.

To claim that because there are no definite chemical substances to prove the presence of an intestinal toxemia against the clinical facts that exist constantly around us, is like claiming that such a body as the sun does not exist. The only evidence we have of it is sunshine and its existence is judged by an indirect analysis. Nobody has ever taken a piece of this planet and analyzed it chemically or synthesized its component parts to produce another sun. Yet the whole world in every activity and substance is dependent upon the sun for existence. In the 1924 edition of Osler's "Practice of Medicine," there are fifty-eight diseases the cause and origin of which are unknown. Would it be proper to claim that these diseases and conditions do not exist just because some chemical or biological substance is not provable? Add to these the diseases and conditions in like unknown etiology throughout the various specialties, and the numbers are in the hundreds. For the absurdity of the statements of these critics, what has been stated can be taken as proper answers to the chemists' contention, to which should be added that chemistry as a science and a hand-maid of medicine may some day develop enough to meet a real usefulness in medicine, and when that time comes the toxemias of the human intestine will most definitely be proven by them. Until that happy time comes, the chemists' opinions, with that of those physicians who believe with them, had best be disregarded. The clinical conditions and the patients are there, many of them are a very sorry and complaining lot, and deeper study of these conditions than is now general will more than prove the existence of these conditions and suggest the best methods known today in their diagnosis and treatment.

The next group of critics comprise not a few of our best men in medicine, many of whom occupy prominent positions as teachers and who are good investigators in other directions. The rule held by these men is partly the argument believed by and derived from the chemists, and also from those who believe that the intestinal flora is solely dependent upon the food-stuffs ingested in the way it is changed according to the abundance of protein and carbohydrate intake. To a certain extent this latter is true because there is no doubt that in definite conditions and with distinctive types of diets a change in flora can be accomplished for a short time. But what these men do not seem to know is that in distinct infections this is generally not possible, and if it is accomplished, it cannot be sustained for a practical length of time, rarely longer than thirty days.

Almost all intestinal organisms are facultative, and this facultative ability may and usually does become active again on a diverse type of diet, and if not promptly, always will become so after a few days or weeks of time. A person with an intestinal putrefaction may be made to change an intestinal flora, as well as the chemistry and toxic factors perhaps within the gut, on a carbohydrate or vegetable and fruit diet; but, after a short time the inhibited organisms will exert their facultative abilities and even though one is persistent with this diet, the original putrefaction, chemistry, and toxicity will return.

The same is true in the many different forms of acid fermentation toxemia (the saccharo-butyric class) when placed on a high protein diet. In addition to the physicians who believe the diet to be solely efficient and sufficient, there are not a few sanitarium who follow this belief, teach it to their patients, and employ it as a fundamental treatment. It always is an interesting experience in the many patients I have seen who have been handled and advised in this way, and who have been persistent with dietetic advice on the part of these men and institutions and followed it with conscientious effort, to find them as toxic as could be in spite of the foods taken. My

viewpoint on dieting in the treatment of these conditions will be presented in a later chapter.

I wish here to state that dieting alone for the primary, and many of the secondary toxemias, looks like a good method, but practically is capable of only temporary benefit if successful at all, and mostly is a delusion and in no sense curative throughout the work.

It is strange that the chemists who are engaged in medical affairs and the physicians who have been critics of intestinal toxemias, nevertheless recognize that bacteria can form toxic bodies capable of producing symptoms and conditions in other fields of medicine. None of these antagonize the proven facts on the specificities of organisms and the vast array of facts that immunizing substances can be proven to have been generated by disease and infections in the body. Much of this point of view is today fundamental in medicine and therapeutics. Yet the presence of none of these substances can be proven by chemical analysis, and still the chemists and most all physicians acknowledge them as existent. If this is true with any organism or infection anywhere in the body, why is it not also true with the greatest of all organism-bearers in the body, namely, the intestinal canal?

For the clinician not to employ the indicated antitoxin in diphtheria, tetanus, or rabies, not to employ vaccination against typhoid, variola, etc., or the protective and active types of sera and vaccines in other conditions, would be equivalent to malpractice. This being so, why must the intestinal canal be considered so different, since intestinal toxemias are almost entirely a bacteriological affair? One may therefore take it for granted that chronic intestinal toxemias are definitely clinical conditions, and directly, or in a coöperative sense at least, most important conditions to be considered in the proper handling of many of the diseases and conditions in medicine; and that until this is definitely believed, and better work than has been done thus far by many men and institutions is carried out, the fullest service to the patient will not be accomplished. The experience of the author has proven this so many times during these last thirty years that there is no question about their

importance, and his attitude is definite on the subject no matter what the criticisms may be.

PAST WORK IN STUDY OF INTESTINAL STATES WORTHY OF MENTION.

It is a strange commentary that the worth-while work of many past workers in this subject has received but scant attention on the part of the profession. In the absence of offering anything better, or even as useful, some have been critical of all, and the majority are indifferent or ignorant of what these have been. Instead, the profession has listened and been influenced by medicaments, colonic lavaging, diets, food substances, and especially those which were graphically advertised, and adopted these standards of treatment as sufficient in the subject.

I wish here to give credit to those workers whose past efforts have been worth while even though neglected. With some, in many instances, my own views will differ in kind, scope, deductions or clinical application. With others I firmly agree. Time and experience have required me to modify, add to, or make different conclusions from the many made and suggested by these worth-while workers in medicine. Nevertheless, their work and contributions cannot be neglected, and these men and their work deserve mention here.

EXAMINATION OF THE FUNCTIONS OF THE INTESTINES BY MEANS OF THE TEST-DIET.

Schmidt's Work.—Of interest and the most worth-while application for some understanding of intestinal conditions is the work of Adolph Schmidt, 2d. This author drew attention to the importance of employing test-diets to test the functions. A good pediatricist knows the diet the child is on and examines the recently passed contents of the diapers to note what happened. Yet the average medical man sends casual cold stool and old urine specimens to a laboratory and then undertakes to deduct a diagnosis of the biologic and chemical condition of the bowel from their examinations. What could be more erroneous or more misleading? Casual specimens represent

casual foods taken and different foods can give vastly different types of stools and urines.

Schmidt properly drew attention to the importance of a standard detailed diet for clinical purposes and quantitative analyses. Unfortunately the use of the Schmidt and Strassburger, No. 2 diet, and even that of Schmidt alone, is difficult of application in practical ways, and, instead of using them, all of my own work has been done on a standard plan—which on occasions is modified slightly or reduced in quantity as in the case of a young person or child, or changed to total protein or total carbohydrate as will be mentioned later.

Schmidt then drew attention to the importance of macroscopic and microscopic examinations of the food detritus, mucus, crystals, etc., all of which are important. His work on the bacteriologic examination is worthless. He tried to simplify this with his "incubator test" so as to diagnose the normal, the gut of carbohydrate fermentation, and that of putrefaction.

There is a certain amount of practical application toward diagnosis in the "incubator test" and it is far better than nothing at all or the usual laboratory examinations that are made; but, for doing proper justice to these cases, the "incubator test" fails too often to be of much use. However, it could be performed in the simple routine examination of stools, especially when media are employed, because it occasionally helps for understanding of clinical types. In no sense can it be depended upon for bacteriological examination, which is the most important of all.

Many additional points were brought out in his small book which is well worth reading. Among these is his attitude on gastrogenic, hepatogenic, pancreatogenic, organic, catarrhal, atrophic, functional, absorptive and motor states. Many veins of theory and assumption weave and spread rather widely through these, and some of his assumptions and conclusions cannot be proven in practice and experience. The work, however, deserves study, but his methods and beliefs not too close an application.

Work of Metchnikoff.—Metchnikoff's work, seemingly ample in biological study, is pointless and useless in clinical ways. The term, "wild races" of bacteria, without there having been the study or even the identification of any, even in groups of organisms concerned, is more sensational than scientific, and his over-assumptive and unwarranted beliefs in the use of the now so-called *B. bulgaricus* should only be recorded as one of the milestones of the past.

Herter's Investigations.—On the other hand, C. A. Herter carried out investigations which are the most prominent and worth while of all the steady workers in this field. In the preface of his book there occur these statements: "The book does not aim at a systematic discussion of the extended and somewhat confused field of gastro-enteric infection, either from a clinical or a bacteriological standpoint. . . . I have laid considerable stress on methods developed in my laboratory with a view of obtaining a better insight into the bacterial conditions of the digestive tract than has been hitherto possible. This I have done in the belief that when these methods are utilized in practice they will prove of real service in gaining a truer conception of the nature of the bacterial processes that are operative in disease. . . . I am confident that the painstaking application of these methods will furnish practitioners with new and reliable indications as to the progress of many cases of infection of the digestive tract."

Since these words were first read (1907) the profession has signally and constantly failed to adopt these simple teachings or apply even his limited (although useful to some degree) insight "in the bacterial conditions of the digestive tract" and the "conception of the nature of bacterial processes that are operative in disease." The only excuse the profession can offer is that the work of Herter was too technical for understanding by the men of medicine, and because they did not understand it, it must be neglected. Yet, here is the man whose work put the subject for the first time to some degree on a scientific basis, and as far as I am aware, no one has taken it up and followed it out in study, research and practice. Instead, Herter's efforts were passed by and the old teachings of

the French workers (colonic irrigations, lactic and ferments, medicaments, etc.) were persisted in. Had the studies of this subject started with Herter's work and the French workers never touched it, far more real advancement would have been made.

It is not the province of the author to deal at length with the real information contained in this small volume of Herter. Some of his points, with added material, will occur in the subsequent pages. But, since this work by Herter covers much that is of most essential value for the understanding of bacterial infections and the intoxications arising from them, the reader of this present book should read the one by Herter as helpful to the understanding of this one. Unless he does this he will meet with omissions in mine that may make for difficulty of understanding matters well presented and now over twenty years old that need not be recorded again. The reason this is important is because practically all of the clinical importance of the subject of intestinal toxemia is a bacterial or biologic one, and this work in large part takes these up where Herter left off.

Other Biological Workers.—It is now a pleasure to record the names of different workers on this most important biological side of the subject. This is presented as an honor roll with the hope that the serious student of this field of work may know the names of those who have rendered signal, although too often forgotten, service; names which could lead them into past literature much of which will not be recorded in these pages, and yet practically all of which is most valuable collateral knowledge for complete understanding. Prominent among these (although there are others, doubtless, whose names have slipped my memory) are the following:

Andrews	Bergh	Bodkin
Archard	Bergman	Boycott
Andrewes	Betz	Braudat
Bail	Bienstock	Brieger
Baldwin	Bjeloussow	Bryant
Bauer	Blair	Bovet
Baumann	Blumenthal	Brion
Baumstark	Bocai	Bensaud
Beijerink	Booker	Batchlor

Bordet	Howard	Pottein
Clemens	Howland	Passet
Collins	Henrici	Prior
Conradi	Horder	Richards
Cushing	Hottinger	Rettger
Citron	Jaffe	Rodella
Castellani	Kruse	Roget
Chambers	Kurpjuweit	Roos
Cohn	Keman	Rahe
Cuboni	Kedrowski	Roger
Doebert	Klein	Robinson
Dopter	Kohn	Shiga
Dunham	Kayser	Schardinger
Duval	Khouvine	Schottelius
Distaso	Koch	Schottenfroh
De Nobele	Kendall	Schmidt
Debono	Livingston	Straussberger
Dorset	Lee	Schotmüller
Drigalsky	Lembke	Selmi
Eberth	Levin	Senator
Eberle	Libbman	Simon
Ehrlich	Lebenan	Smith
Ellinger	Lesage	Stefanio
Emminghaus	Loeffler	Stokvis
Escherich	Lebenau	Schroeter
Emmerich	Malzushita	Sternberg
Flexner	Maurea	Thierfelder
Friedländer	Maury	Talma
Ficker	Manteufel	Taylor
Flügge	Morgagni	Tissier
Foster	Martin	Torrey
Fraenkel	Mereschkowsky	Trevisan
Ford	Metchnikoff	Udransky
Freudenreich	Meyer	Uptadel
Finkler	Moro	Van Ermengen
Gaffsky	Murath	Veillon
Gartner	Müller	Vedder
Gram	Miller	Welch
Grassberger	Nencki	Wakeman
Germano	Neubauer	Ward
Gaertner	Nuttall	Wassermann
Gessner	Oebius	Werner
Garnier	Park	Willimsky
Gengou	Passini	Wollstein
Herter	Pasteur	Wright
Hervieux	Petruschky	Wilson
Hilgerman	Pfeiffer	Zuber
Hiss	Pezenti	Zinsser
Hopkins	Porcher	Zopf

THE INTESTINAL TRACT AS A SOURCE OF OTHER FOCAL INFECTIONS.

When all has been said about apical, tonsillar, sinus, prostate, female pelvic organs and other commonly recognized focal infections, the intestinal canal outranks them all in importance. This is not recognized because the average medical man knows little about it, and the specialist nothing at all. When about one-third of the bulk and weight of dried feces is solid bacteria which are voided in countless trillions in one day, and when the organisms in many of the infections met with throughout the body are those of intestinal origin, one would think that the intestinal canal should receive more attention than is being paid to it.

Where do the *Escherichia coli* in a pus kidney come from? Where do the organisms of an infected appendix come from? Where do the organisms of the gall-bladder arise? A pelvic abscess, liver abscess, or an abscess most anywhere in the internal structures, is only rarely caused by infections from the head or chest, and practically always from the intestines. What makes adhesions in the peritoneum, and when there are mixed infections anywhere throughout the body from whence these added organisms?

The defensive structures to their introduction are the disintegrating and lytic processes within the bowel, the mucous membrane of the intestines, the bactericidal powers of the liver and the phagocytes. May these not break down and then exit from the bowel, as proven when we can so often culture intestinal organisms from blood in the veins? From whence come the organisms in the veins of phlebitis, lymphangitis of the internal structures, and lymphadenitis? The skin enters some, the head and throat structures others, but compared to them the intestinal canal is like Broadway beside a small alley in Greenwich Village. Not all tooth infections are due to the *Streptococcus viridans* and even if they were, this organism is a common denizen of the intestine, culturable from the stool in the same person, and it does not disappear from the bowel when the teeth are removed. May they not go up from below

as well as come down from above? Where do they start from to get into the teeth?

Recently as a result of bacteriological examination of 192 teeth in 74 patients, this organism was not met with oftener than in 15 per cent. of these infections. The hemolytic, non-hemolytic and the *Streptococcus fecalis* were found instead and these live in the intestines like innocent and inoffensive people in an apartment house, and there are lots of apartment houses in which they live. Like people in such houses they occasionally go out and when they do, they stay out, live on a tooth, in a tonsil, in a gall-bladder or somewhere else, and when locked up in a tight place they become criminals. But they started from the intestines. They are now a focal infection in a tooth, tonsil, etc., but they were a focal infection in the intestine formerly, and my experience on this point is so conclusive that debate and question are no longer tenable with me.

In 500 vein taps made for Kahn and Wassermann tests on office patients, none of the persons especially ill, positive blood cultures of intestinal organisms were met with in almost 6 per cent. Had more than one tap been made on the one person at different times, this percentage would have been higher. This proves that commonly intestinal organisms gain entrance into the general blood stream and do no apparent harm at the time, but they at the same time could easily cause focal infections in various parts of the body. These organisms are generally of a low order of virulence and their infections would cause chronic types of infection rather than the acute ones, although the latter could occur.

But, so far as the intestine as a source of focal infection is concerned, there is in this matter a far more important subject, and that is the intestine as a residence of focal infection and what toxemias from this canal mean in the production of diseases and conditions of the body. No physician, general or specialist, can give full service to ill people in practically all chronic diseases and disorders without having to give a full consideration to the biology of the intestinal canal. Organisms in themselves do not produce much if any baneful effect on the tissues of the body. They may in septic processes

locally, but the real hit most commonly comes from the toxins they produce and all of these are also resorbable from the intestinal canal. From many angles this has been a debatable subject in medicine since Bouchard's day, but in the light of my experience, beliefs, and clinical results, the broad hand of fact grips the whole work so fixedly and so significantly that there can be no question about it.

The intestine as a source of focal infection is not a theory, not an hypothesis, nor is it just a dietetic or elimination matter, it is not a rest, medicament or a colonic irrigation affair, and it goes beyond things done at foreign spas, or the taking of *B. bulgaricus* or *B. acidophilus* by mouth. Instead of these makeshift methods of treatment, it is a deep biological problem, inconclusive in places, indefinite in parts, difficult to describe and ever shifting and variable, and in it are the greatest lengths of time and extents of experience and of laboratory procedures for understanding diagnoses and treatment required, as well as constant searching, researching, checking and rechecking. At no time is it what all of us have been taught as medicine, namely, the making of a diagnosis in all that this may mean, a treatment, and a happy expectation of a cure or a worth-while result; it is not as simple as that, but rather a difficult procedure and one in which experience is the only real teacher. This looks hopeless no doubt to some, but medicine in the future is going to be far from as easy a process for diagnosis and treatment as it is today. Constantly it is getting deeper, spreading out farther and going into biology and other fields that look super to many of us today.

But with all there has been up to the present, we really have only scratched the surface of what will be the proper handling of the sick in a few years to come. How wonderful this future of medicine is going to be, how interesting it already is, and how much satisfaction has it given to me that the errors of intestinal biology have figured in cures and amelioration of so many obscure conditions! I wish definitely to record, that the intestinal canal is the most important source of focal infection in the body, and its biologic disturbances are the greatest and most important causes of toxic

factors in the human being. These facts are not stated as a guess, a theory, or an hypothesis. They are provable laboratory and clinical facts.

Up to today 181 different organisms from the human intestinal canal have been identified. Each of these is a distinct genus, so far as we can say at the present time. To these no doubt a good many more organisms will be added, such as those now found in soil, water and in connection with various foods that gain entrance into the human digestive canal, and which will be discovered in the intestines in the years to come. Of these 181, 72 have been proven to be definitely pathologic and were only discovered in connection with infectious and diseased conditions that they brought about.

There are 50 saprophytic organisms—microorganisms that live on dead tissues and food substances, practically all of which are disease and toxin producers in the human body. Of the zymogenic forms there are 49, not a few of which are most inimical to carry in the intestinal canal. There are but 10 more, which, so far as we know, are non-pathogenic. Of the pathogenic, and somewhat of the saprophytic and zymogenic types, there are many strains. In the *Escherichia coli* group alone there are 21 different strains, some of which are harmful and others not so much so, and some not at all. One can now get somewhat of an idea of what a laboratory of infection the human intestinal canal really is and the rôle it plays in focal infections. It is etiologically, clinically and therapeutically so important, that when we talk about focal infections (in the most extreme statements that can be made about teeth, sinuses, tonsils, etc.) we today have to look to the intestines as the main factor of the whole subject of focal infections as well as the main biologic factor in most investigations that pertain to toxemias.

CHAPTER III.

HISTORY AND SYMPTOMS OF THE PATIENT.

During undergraduate years the medical student is taught internal medicine under the classifications of "Etiology," "Pathology," "Symptomatology," "Diagnosis" and "Treatment." Sometimes additional data is presented under "General Remarks," "Differential Diagnosis," "Complications," "Laboratory," "X-rays," and the treatment divided under "Medical" and "Surgical."

After graduation and during interneship or when in practice for himself, the doctor begins to forget all of the less prominent shades of color in these pictures and his interest and attention become more centralized on certain prominent and outstanding parts of a disease or disorder. With sufficient hospital or clinic practice there builds up in him an understanding of this or that disease which he applies to practice in the readily understandable or remembered points. As is most proper, he soon begins to apply the pivotal or characteristic symptoms or syndromes (as they are called in later years when one or more are conjoined).

So far as suggestions from the taking of a history are concerned, most all practitioners listen to or undertake to elicit this or that complaint of the patient, which now become symptoms. These being added up and fitted together, lead his mind to certain probabilities or possibilities. In addition to the subjective story on the part of the patient the doctor then undertakes to discover objective findings (more symptoms) by physical examination; and by this process, to which may be added laboratory and x-ray examinations, a diagnosis usually is (and should be) made or attempted. By this sort of instruction of the mind and practice in diseases and disorders, one has by extensive and intensive study over years of time and devotion to their practical application the safest route to the making of a good diagnosis, and thus a good physician. To him then disorders and diseases stand out in certain classic

and individual ways, and as the various cases match up in this or that form and locality of the body, accurate diagnoses are made more easily and in higher percentages.

DIAGNOSTIC DIFFICULTIES IN INTESTINAL TOXEMIAS.

In intestinal toxemias none of the above mentioned fundamentals of clinical medicine, understanding, diagnosis or treatment can be utilized, and this, as much as anything else, has developed the thought in many otherwise good medical men that conditions such as intestinal toxemia do not exist. It is almost impossible for many men to get beyond the restrictions of their fundamental training, and for the majority of men in medicine to believe a disorder exists when there are no classic symptoms of it. Practically all of these same men realize that intestinal disorders of toxic states exist because they could not fairly interpret many of the disorders of patients in any other way, but beyond that belief or assumption they cannot go, and the majority of these are not interested enough to conduct investigative work to find out whether such is the case or not.

It must be stated definitely that there are no classic, individualistic or pathognomonic symptoms of intestinal toxemia. Diagnoses are made by suspecting its presence, and then utilizing the laboratory and engaging in study to prove or disprove its presence is the only way possible. It, therefore, seems to be unnecessary and even improper to give a narration of symptoms. Further, this would not only be unfair to the subject, but might be misleading and harmful.

A diagnosis of intestinal toxemia might be made by them and far more important conditions left unsuspected and undiscovered. One needs here to clearly bring out to the reader and most definitely impress upon his mind what may be termed classic symptoms, for there is no abdominal condition, malignant, organic, neurotic, infective or any other, in which all of like symptoms do not run through the whole gamut. It is true that certain disorders like gastroduodenal ulcer, appendicitis, intestinal obstruction, ulcerative processes, etc., may

have quite a classic symptom-complex or sharp anatomical localization, but even here the same symptoms of pain, distress, gas, belching, constipation, etc., run through them as they do in all the others which are minor.

In even the most definitely fatal disorder—cancer—there are usually no symptoms until the growth is well advanced, too late for cure. This cardinal fact of common absence of definite symptoms, or the colloquial forms present throughout all abdominal diseases and disorders, must not be lost sight of, and, as stated before, it is also only a short-sighted type of medical man who claims, because there are no characteristic symptoms, that intestinal toxemia does not exist. Innumerable times, ulcer, appendicitis, gall-stones and other diseases exist with no definite symptoms, and in not a small percentage of them all there are no symptoms whatever.

The abdomen is a cavity of the body in which the contained organs are peculiarly bereft of the production of symptoms even when badly diseased. In this way it is unlike the cranial cavity and that of the thorax, the latter being in the middle ground between the cranial cavity on the one hand and the abdomen on the other.

In a subsequent chapter disorders are mentioned in which investigation should be conducted as to the possibility of a causative or relative chronic toxic state in the intestines. While in any of the disorders mentioned this is important, attention is sometimes drawn to digestive disorders which are caused by and bound up in this significance. Unfortunately this is not recognized even by gastro-enterologists, this being so because of their lack of knowledge of the significance, study and experience in these states.

Throughout a mass of writings on digestive states, hardly a word appears on toxic intestinal states, and if so it is most cursory. Yet the fact remains that these intestinal states are a far more worthy, practical and accurate explanation of many of the conditions described than the theoretical explanations made. Part of this lack of knowledge is due to the fact that the gastro-enterologists in the past and present have paid too much attention to the stomach (a relatively insignificant part

of the digestive tract) and not enough, if any, to the great digestive laboratory of the human body, namely, the intestines. Woven all through my experience are innumerable cases of liver and gall-tract, secretory, motor and sensory stomach disorders, neurotic, nutritional, stases (gastric, ileo-colonic and straight colonic), appendix, colitis, adhesions, ptosis and other strictly gastro-enteric states, the permanent cure of which was brought about by the cure of the toxic gut condition alone.

More liberal in this connection, but not more able in service rendered, have been our so-called internists. With them, toxemias are more generally recognized and not uncommonly handled with dietetic, physical or medicinal efforts to control them. In this sense not a few of our internists have rendered a gastro-enteric service superior to gastro-enterologists. But in my observation the type of worker in medicine who appreciates those intestinal states the keenest are those who are engaged in the more sharply limited specialties, like those devoting their time and attention to skin, nose and throat, eye, nervous disorders, joint conditions, etc.

Etiology in their cases is often vague and unknown. Administrations are made to case after case that comes to them, local relief and benefits are accomplished, but commonly they feel that the fundamental cause is receiving no specific attention. Purgatives, tonics, diets, rhubarb and soda mixture, colonic irrigations is the armamentarium among them, which commonly they can prescribe and conduct as well and sometimes better than do the gastro-enterologists, or the internists, the two classes of men they have a right to look to for better types of investigation and treatment.

Thus numerous men in minor specialties of medicine today are looking for better work in this connection. Attention to focal infections, which they are all most keen about, has helped them considerably, but there still remain the larger number of these patients in whom the etiological work is negative. What happens then? They repair to the innumerable laboratories for examinations of specimens of stool, urine, blood, etc.

As a rule the laboratory helps but little more than gastro-enterologists or internists can do. But at least fundamentally

such doctors show their appreciation of the significance of these states far more than the gastro-enterologists, the non-biologic types of medical man, and the general surgeon. There is no doubt that it is in this work we gastro-enterologists and internists can give these men a service far more worth while, giving to internal medicine another great service from gastro-enterology, and filling a gap, which on the whole, is worth all the study and time it takes.

As practical experience in medicine increases, the suggestion of the presence of an intestinal toxemia commonly presents itself. It is largely cardinalized in the chronicity of the complaint in which many different diagnoses and treatments have been made and carried out, the whole course interspersed with intermittent periods. Involved in this are a large number of complaints which run from the head to the toes.

Sometimes they group themselves up in locations of the body such as the head, chest or abdomen; sometimes they are scattered throughout the body, and may even comprise only one or two symptoms—all else being negative. In this irregularity the medical man must definitely learn or unlearn an important fact, and it is as he accomplishes this, that his vision and understanding will be clearer. For years we have been taught to accept the writings and beliefs of workers in certain fields of medicine who have attempted to teach us new entities or new aspects of old things.

For a number of years there has been an abundance of literature leading to an endocrine explanation of many complaints. I do not refer to disorders which we all agree are endocrine in origin and which can be provable as such. But I refer to the vaguely understood matters which enthusiasts in this therapy would have us believe were due to or cured by these inclusions. While little is definite in this whole field of work, nevertheless imagination and enthusiasm have strung (so-called) endocrinology through a large amount of clinical matter, way beyond the real values of this therapy.

Endocrine treatments are used for almost everything clinical that is not understood, occasionally with benefit, but usually with none. This goes through from the growing

child to the grave of the aged in a large number of clinical states or complaints. While here and there is an instance where it has been helpful, the great majority of failures eclipse it almost to a folly and a waste of time, money and effort. The vast majority of these cases are due to chronic intestinal toxemia, and while some may have an endocrine aspect, this usually has been brought about by the gut toxemias having a depressing effect on the glands of internal secretion. This erroneous idea also goes through other departments of interest in medicine such as allergy, protein sensitization work, general biological work, etc.

A COMPILATION OF SYMPTOMS IN INTESTINAL TOXEMIA.

Repeating a statement made before, namely, that *there are no definite symptoms of intestinal toxemia*, the author appreciates that with not a few of the readers of this work many would still be influenced by the old standards of medicine, namely, interpreting symptoms for diagnosis. While the author has long ceased to attempt more than a suggestion from symptoms, and believes that their interpretation of an intestinal toxemia diagnosis is of little value, he still herewith presents a compilation of the symptoms noted in the histories of five thousand cases of both the primary and secondary forms of intestinal toxemia.

Perhaps the reader can gain instruction from these that would help him; I certainly could obtain only a suggestion in a history sense. If others are like myself the narration could at least prove the protean nature of these disorders, and the significance of intestinal toxemia as almost a blanket-broad clinical condition.

The subject of chronic intestinal toxemia is bound up in so many vague clinical conditions that to describe the history of these cases in definite ways is impossible. On one hand they may be outlined about as broadly as is the field of medicine and on the other they may be described in a compilation symptom sense. In this connection it is not the purpose to present them from the laboratory or x-ray standpoint. An

attempt will be made only to classify the symptoms in a semblance of order under subjective and objective arrangement.

Fatigue.—Fatigue in the fullest sense is a relative or individual matter. There are people who seem to be born tired, people who tire relatively more easily than others with the same degree and kinds of application, and still others who tire easily on mental strain and feel this physically when really they are sufficiently rested. One must, therefore, judge the individual in work, application of mind, age, hours of sleep habitually taken, and, if there has been a period of time in their adult years that they were not fatigued and now, for no evident reason, this fatigue is present in a steady way. My belief is that the symptom of fatigue, which is a prominent one in intestinal toxemias, should be closely analyzed and every reason for it be searched before it is recorded as a symptom of toxemia. For it to be definite for toxemia, it must have been acquired (unless the toxemia can be judged to have started in the “teens” or childhood) in adult life, be continuous, and to have been completely relieved by the treatment of the case.

Fatigue is a symptom running rather generally throughout many disorders and diseases, but that which is so prominently mentioned among endocrinologists in connection with gland deficiencies is more often due to intestinal toxemia than hypofunction of the internal secretion glands. One needs only to carefully analyze enough cases of so-called gland deficiencies or dysfunction to note that the best service of the endocrinologist is not to “feed” glandular substance, but to treat the intestinal toxemias which have caused the dysfunction of the glands. Where is the logic of feeding gland substances for deficiency when the deficiency is due to depressing toxic effects on the glands coming constantly from the bowel? This is the reason of failure that one so often meets with in glandular therapy, either because the glands are really not deficient and the symptoms are due to the toxemia itself, or if deficient, glandular feedings will not fill the gap, the toxemia still continuously operating on the gland in spite of them. This has been mentioned before but cannot be over emphasized.

Definite symptoms of fatigue are conspicuous in intestinal toxemia, and many of these individuals engaging in moderate amounts of work and mental effort become quite exhausted and must rest awhile before they can recover their sense of well being, which is easily exhausted again. With this may be more or less pain in the occipital region, in the back and in the legs, with perhaps vasomotor disturbances at times. As a class, such persons not only seem fatigued on exertion but have a constant-running fatigue which when prominent makes them irritable, cross, and affects them more or less mentally through loss of mental control and even disposition. They may look well and yet can stand but little. One sees this in the financier, teacher, farmer or business man who easily becomes irritable and cross, who fatigues at his work early in the day or is "all in" at week-ends, as well as in women in all stages and conditions of life.

Not infrequently conditions become so bad that complete rest, vacations and sharply modified existences are essential for continuation. I hold to the standard that a person, well physically and chemically, should be able to stand the "daily job," any kind of worry in business, domestic and social life without effect on efficiency, feeling of well being, or change in disposition. This is a high standard to set, and occasionally one has to modify it, but as a rule it is the proper standard to adhere to, and, if the condition is not explainable on some other basis, an intestinal toxemia should be suspected and examined for.

Fatigue, carefully analyzed and judged, was found as a symptom in 70 per cent. of my five thousand cases. Often other symptoms due to fatigue were also present, such as dizziness, indigestion, aches and pains, slight nausea, insomnia, lack of interest, etc.

Insomnia.—Insomnia is a common symptom of intestinal toxemia, but it must be remembered that it is not a characteristic symptom of any one disease or condition, but is common to many. We are all aware of the sleeplessness during the night that so many of our ill patients suffer from. In our hospitals the wards are largely "awake at night." Pain

and distress are commonly the cause of this, but the toxic factors of illness, with or without fever, are also important. Insomnia as a symptom in intestinal toxemia belongs to this latter class, and it is often surprising to note what large amounts of hypnotic and soporific drugs are employed to master it, no attention being paid to the etiologic intestinal condition.

Generally these individuals, who have eaten three meals in the day, are considerably more toxic at the end of the day than in the morning, and it is not uncommon for them to be awake in the early part of the night, getting asleep when quite exhausted along toward the small hours of the morning, while in the morning they are quite sleepy and it is difficult for them to arise. The urine at the time of the insomnia is often high in preformed sulphate bodies and other toxic substances absorbed from the intestinal canal, whereas the urine formed during the day is relatively more normal. It is suggested that these toxic substances stimulate the central nervous system, making sleep difficult.

Another factor of the toxemia involved in the production of the insomnia is the effect of the intestinal toxemia on the nervous system. One has here, of course, the fundamentally under-endowed individual, neurasthenic, psychasthenic, the neurotic and others, and it is in such individuals that the toxemia is a distinctive factor, in a sense intensifying the symptoms, deepening them or bringing them out in a more pronounced form. In addition, one has to contend with individuals whose insomnia is the development of bad sleeping habits—those who have used the bed at night as a place to engage in thoughts, reading, etc., and that interesting group of people who have never acquired the ability of negating the mind in a static way to produce by mental blankness and general body relaxation the induction phase of sleep.

Everybody has this acquisition of a physiological induction to sleep in a more or less degree, but when it is poor, quite naturally an intestinal toxemia, by keeping up an activity in the mind, makes the combination a distressing one for many people.

Anemia.—Not uncommonly a moderate degree of chronic anemia is present. Usually this is of the simple type, but at times, especially in *Cl. putrificus* infections, it is of the pernicious form. This anemia is due to resorption of sulphur products, mostly sulphurated hydrogen from the bowel, these substances hemolyzing the erythrocytes.

Oftener again any of the hemolyzing organisms that can affect the blood by their toxic products from the bowel are the cause. With them one may have disturbances in menstruation, either painful, scanty or profuse, these being due to the anemia or toxic depressing effects upon the ovaries or the thyroid.

Tendency to "Taking Colds."—With those of sedentary lives and types, especially when housed up during cold weather in poorly ventilated rooms, having insufficient exercise and fresh air, the anemia might be more pronounced, and they be more liable to colds and respiratory infections. The main reason for these infections in the late winter and early spring is because a steadily running intestinal toxemia had gotten the upper hand of vitality and resistance. There is little doubt about this because one sees more effects from intestinal toxemia as the winter progresses. After the winter, in the temperate zones, in the late summer, fall and early winter the symptoms are distinctly less. The reason for this is that while their toxemias are running constantly, fresh air, sunshine, fresh foods and exercise, which are part of daily living in the more pleasant and open seasons, bind the toxic products in the blood and body, at which period the symptoms are relieved.

Various studies have been made and scientific commissions have worked on the study of the causes of the "common cold." None of them will succeed in the solution of this question until the biology of the gut is estimated as one of the main contributing causes. All of the organisms isolated thus far from nose and throat regions are those present around us at all times of the year, summer as well as winter. The fertile soil for their growth is the toxic body, continuing toxic over weeks of time in cold weather, from organisms originating in an intestinal canal that harbors a biologic toxemia. Common

colds, pneumonia and influenza had become markedly less existent in patients in whom it had been possible to accomplish and maintain a normal biologic intestine. This sounds like an extreme statement, but it is true.

Anorexia, Fitful and Intermittent Appetite.—These are occasionally complained of by patients rarely having a sharp appetite and desire for food. They often go along for days eating moderate amounts of food in a more or less indifferent way. Often on very moderate amounts of food they are distressed with weight and pressure in the epigastrium, which may or may not be relieved by belching, increased gas in the stomach being a common symptom. Usually this is temporarily relieved by purgation. Whatever the treatments may be, a loss of weight is commonly the result, especially in the ptotic individual in whom often one of these bowel conditions is the main cause of symptom relapses. At other times they may eat well for a few meals and then suddenly the appetite will drop off again. Or they may eat well for a week or two and the same thing happen. The appetite is taken away when the toxemia is at a high mark, to be restored when it is lower.

Most intestinal toxemias run a cyclic or intermittent course, few of them being constant and steady in symptoms produced from them. Anorexia as a symptom is an excellent index of this, so much so that when a steady good appetite is present one can safely look for digestive troubles from another cause. Fear of taking foods on account of distress should not be confused with anorexia as a symptom. This symptom of anorexia and fitful appetite, more or less pronounced, is present in 34 per cent. of cases. Vomiting is almost nil.

Definite Gastric Distress.—A most common symptom in the gastric type of case is pyrosis. Distinct symptoms of hyperacidity may be present, yet many times the test meals do not prove this existent and still the patient complains of "burning in the stomach" after taking foods. Oftener this is classic of hyperacidity, namely coming on one or two hours after meals, and relieved by alkalis. With these the test meals will commonly show a high acid index or curve.

One should always suspect the complaint of pyrosis, where there is a normal or acid-low test meal, especially if not relieved by alkalis, to be one of intestinal toxemia. And the same is true of the hyperacidities in which the usual causes for this are not present (faulty and over-abundant eating, vagotonia, ulcer, etc.). This symptom is due to the intestinal canal being acid above normal and the symptoms in the stomach being an expression of or a concomitant state from this. Pyrosis, intermittent or more steadily present, was present in 23 per cent of the cases, and sharp attacks of pain due to pylorospasm or gastrospasm without definite lesion in 11 per cent.

There may be distress on taking food due to an increased sensitiveness in the stomach, a hyperesthesia gastrica. This is due to irritation of sensory nerves from toxic products. Most of the achylas, as Faber has shown, are due to granular types of chronic gastritis caused by chronic intestinal toxemias.

Gas in the Bowels.—It should be recorded that as one gets older one has more gas in the bowels. It may be debated whether this should be judged as normal or not. In my opinion it is partly normal, but most often it is due to the gradual onset of an intestinal toxemia as the years have advanced. Where this is definitely present short of or continued from before the fourteenth year, it is a symptom of note, the same being true when excessive gas formation—in attacks with distress or continuous without distress—is a complaint of the older patient.

In the analysis of the two, the young or middle-aged person with much gas, and the very old one with hardly any, the young and middle-aged has an intestinal toxemia, and the old one perhaps a more normal bacteriology. This has been proven uniformly hundreds of times. The truth is I have never seen a person over eighty years of age who did not have a non-toxic bowel, and because of this I am inclined to agree with Metchnikoff in that long life is dependent more upon a normal biology of the intestinal canal than any other one factor.

Of course, one must be analytical enough to figure out whether increased gas in the bowel is positive or only assumed

without good reasoning or knowledge, and also to figure other factors in which this may occur as a symptom.

There is a characteristic odor to the stools in certain types of toxemias which, while not always present, is frequent. The putrefactive type from sulphide formation is accompanied with large quantities of sulphureted hydrogen gas giving to both stools and the gases from the bowel a putrid odor. Gases passed in this type are characteristic. Fortunately in the putrefactive type the gas formation is not as abundant as it is in the fermentative types of the disorder. In these the gases are heavily laden with the volatile gases of fermentation (butyric, acetic, etc.), these giving to stools and the gases voided a sour or stale odor.

Then there are other more definite infections where the gases may preponderate one way or another, and perhaps even change in character of odor from time to time. Again in specific types of infection the gases voided may not be especially offensive, being largely of carbon dioxide and hydrogen. In these the gases may have been manufactured within the gut but most times have been passed into it from the blood, the infection causing a neurological or vasomotor phenomenon in the bowel wall function allowing this to occur. There are certain definite infections, such as those due to the *Cl. welchii*, *B. histolyticus*, and some of the saprophytes, especially of the Gram-negative groupings, where an abundance of gas is characteristic.

It must be evident, in people who are troubled continuously with gas in the bowels, causing either distention or frequent voidance, that these are very liable to be instances of toxemia, and in this sense the symptoms are of significance.

Constipation.—There are many reasons for habitual constipation. Most of the causes are found in faulty diets and sedentary habits, and carelessness, ignorance and false modesty are causes in not a few. In the child and the aged, relatively increased absorptive and atonic states and deficiency of peristalsis figure. To these, of course, are added the instances of conditions in which constipation is a symptom, such as colitis sicca, spasticity of the colon, malposition and flexure

defects, strictures, hypertrophy of the O'Beirne or Houston valves, the levator ani muscles, the anal sphincter (also spasm), atonic rectum and others. Of these no description need be given here.

I am in no sense in total agreement with Alvarez that constipation is of little significance in medicine. There is no doubt that many people may be markedly constipated and still be in good health, but not all of such remain so for great lengths of time. And there is also no doubt that not a few people have become "hipped" on the importance of daily evacuations from the bowel, and believe themselves ill if such do not occur. But against the small number of these is the greater number who must have daily evacuations or they are moderately ill, and usually these are instances of chronic intestinal toxemia.

There has been much debate on constipation carried on since Egyptian days of medicine, and it still continues. There seems to be no uniformity of opinion in medicine of what constipation really is—when a person is constipated and when not so. We have the Alvarez idea on the one hand, and Kellogg's, of three stools a day, on the other. It is manifest that both are both wrong and right, it being entirely a matter of the individual person as to the presence and the degree of toxemia also existent. If one is not toxic, constipation is of little importance. If toxic, more frequent movements are required for health and well being. Nobody can answer this as well as the person himself, those being truly "hipped" on the matter being relegated out.

As to movements themselves, it may be said that those whose bowels move daily at about the same time, whose feces are constantly soft (or if cylindrical the last part being soft and tapering), and in whom, without taking purgative or laxative measures, a feeling of emptying or satiety follows evacuation—those persons are not constipated.

But as edema or a cough is only a symptom, so chronic constipation may be a symptom of intestinal toxemia. The irregularity of bowel movements is caused by certain factors that are operative steadily. The most important of these are the granular degenerations in the plexus of Auerbach and

Meissner and the fibers and ganglia of the sympathetic plexuses. These occurring, the normal function of the bowel is interfered with by being "cut off" in its normal innervation. These degenerations are caused by resorption of toxic products from the bowel. Another and still more important result is the constipation that ensues from degeneration and necrosis in the intestinal mucosa, this being especially manifest in the lymphoid tissues and the small glands as well as in the epithelium and secreting cells of the tubular glands. Sometimes all of these elements are badly diseased or destroyed, the process extending down into the submucosa and lymph nodes. All shades and kinds of such pathology are commonly found at autopsy in bowel sections, generally irregularly distributed in the colon, rectum and lower ileum. They are due to toxic products manufactured in the bowel which have caused lysis and degeneration of these structures, and, when the condition is established, constipation is generally present.

It is unfortunate that the profession does not recognize these facts, because this type of constipation (which is very common) could often have been stopped before these destructions occurred. There are no simple clinical indices that this is ensuing excepting the late ones in the x-ray findings of colonic stasis, spasticities, etc. This cause of constipation often starts in childhood and early adult life, advances slowly and insidiously, and is made viciously worse by purgative laxative measures and even enemas. It is sad here that medical men have allowed these types to run on until one sees so commonly the markedly advanced "colon wrecks" for which little in the way of complete cure could be accomplished.

Not infrequently, however, by the proper handling of the intestinal toxemia, these pathologic states reconstruct themselves to normal. There is no doubt that almost all the instances of colonic stasis and spastic constipation are instances of intestinal toxemia in which more or less pathological change is present, and it is an indictment of the profession if allowed to continue through unintelligent methods of treatment. Every person who is constipated is worthy of a complete examination for the cause, and if it is secondary to a toxemia, should

be properly handled and not just fed with laxative diets, given medicaments, mineral oil, lavages, etc.

Diarrhea.—Chronic diarrhea is not a primary symptom of intestinal toxemia, but it not uncommonly occurs as a secondary symptom. Dysentery due to the various forms of dysentery-producing organisms, amebic, luetic, cancerous and the tubercular forms as well as those due to gastrogenic, neurogenic and the rare dietetic forms, will not be considered. There are, however, bacillary forms due to infections of the various kinds of streptococci, parasitic *Escherichia coli*, etc. These are commonly accompanied by congestion, inflammation and ulceration, and are designated as ulcerative colitis (see Colitis). The latter are instances of infection of the bowel contents and gut wall and properly belong to toxic gut conditions, especially in an etiological sense.

Not all of the gastrogenic forms of diarrhea are due to achylia. Definite distinction is always difficult here because a chronic intestinal toxemia may bring on an achylia secondarily, and it often is impossible to separate cause from effect. One must not leave out of consideration the diarrhea that accompanies gastroduodenitic states, and especially those due to the gut contents being so irritating that stimulation of frequent movements (generally not distinctly diarrhea) is the rule. It is difficult, too, to separate the neurogenic and dietetic causes from intestinal toxemia *per se*. All of these may exist more or less complexed in the individual case.

Regarding the main cause we are liable to believe it to be neurogenic or dietetic, while the cardinal condition is that of intestinal toxemia. These are mainly instances in the saccharobutyric groupings of intestinal toxemia in which distinct looseness of bowels with occasional diarrhea occurs. In fact, in these types constipation as a symptom is rarely present. If the irritation of the bowel is marked, and the spastic feature absent, diarrhea may be a symptom of clinical significance.

Colitis.—A symptom often mentioned by patients is increased discharge of mucus from the bowel, either with the stool or passed as such. If it is from high in the colon it rolls itself in condensed form and passes as "shreds," "long strings,"

etc. If from low down the mucus is gelatinous in character. The first is usually passed with stool, the latter also, but may be voided without feces.

The profession has been much concerned of late years in estimating the causes of chronic colitis. The most frequent are neurogenic, allergic, endocrine, and several others, and yet nobody seems to have thought or troubled with investigations to note whether or not chronic toxemic states could be operative in this connection.

The writer wishes here to state that the main cause of this condition is that the colitis is a secondary result from long standing and neglected intestinal toxemia in which the discharges of mucus are an effort on the part of the bowel mucosa to protect itself, or that it is a result of a long-standing toxemia in the bowel and, therefore, of pathology. In my observation and experience these are so constantly the facts that while other contributing causes may be looked for, the complaint of the discharge of mucus alone is always symptom enough for careful study of stools. Unless this is done and the cases of colitis are handled better than they are today, colitis will never be understood, treated properly, and will remain in the chronic domain. There is no condition more definite of cure or control than this type of colitis when proper diagnosis and therapy are applied.

The symptom, as reported by patients or found on examination of feces in the laboratory, is present in degrees in at least 80 per cent. and it is found in all types and kinds of intestinal toxemia. It is more often an intermittent symptom, playing off and on in the person according to the status of the toxemia present at the time and the general state of health.

Abdominal Adhesions.—Patients often report that diagnoses of adhesions in the abdomen have been made. With those who have had pelvic or abdominal operations following septic conditions requiring drainage these are to be expected. They are also to be expected from non-infected or “clean operations,” from trauma due to handling of the parts, but these usually are not extensive and rarely productive of symptoms. There are, however, instances of people who have distinct

and even large areas of adhesions who have not nor never had the usual infected states for which operations are performed, these adhesions not being of the embryonic types. Such adhesions are almost always subsequent to intestinal toxemia in which the bowel defenses have broken down and the organisms gaining the peritoneal surface cause inflammation with adhesion formation.

Whenever the cause for abdominal adhesions is not palpably explained by some local septic process (and sometimes in those, too), careful examination of the individual for the presence of an intestinal toxemia is required. With practically all occasions of pericholecystic, perienteric and pericolic adhesions, intestinal toxemia should be suspected unless proven otherwise, which is most rare.

Gall-bladder Conditions.—Chronic cholecystitis, with or without stones, is due to a secondary infection from the bowel in the vast majority of the instances. These infections occur by the bacteria being carried in the portal stream, and, the liver not destroying them, they gain the biliary radicals and infect the bile, and from this, infection of the gall-bladder and the ducts takes place. The occasions in which the gall-bladder and ducts are infected by direct extension up the duct from the bowel or by blood stream route are comparatively rare.

It is an interesting lack in the medical aspects of the diagnosis and treatment of chronic cholecystitis and cholangitis that all interest is centered on the gall-bladder. The diagnosis by x-ray and the duodenal tube, and the treatment by diets, medications, medical biliary drainages, waters, etc., are all centered on a state resulting from intestinal toxemia, and no attention is paid to the latter. It often is fortunate for the profession that patients are so easily "hood-winked" by these treatments.

Where is the logic of centralizing one's attention and interest on the gall-bladder alone, and neglecting the causative condition? Here and there, of course, when the symptoms arise from a definite gall-bladder pathology, relief would come from surgery or treatment devoted to the gall-bladder as such, especially since in so-called "medical biliary drainages" instilla-

tions of magnesium sulphate solution help to clean out the intestines (mainly the small) by purgation and in that way the intestinal toxemia is benefited for the time being. I am distinctly of the opinion that this is the main reason for relief of gall-bladder symptoms from biliary drainages.

It, therefore, is important when in the history of the patient a diseased gall-bladder had been removed, or that a diagnosis of chronic gall-bladder or duct disease had been made by competent men, that intestinal toxemia be suspected, examined for and treated properly. There have been many instances, even when the symptoms were those arising from the condition in the gall-bladder, where the handling of the gut condition alone brought complete and lasting relief in the gall-bladder.

In my opinion, not to remove an intestinal toxemia under a diseased gall-bladder condition is short-sighted medicine. And, further, if an operation had been performed and the gall-bladder found to have been diseased, that is sufficient reason, with a patient still complaining, or even when not, to search the intestine for the original cause of the trouble.

Appendicitis.—What has been said above in connection with gall-bladder conditions also holds true with the appendix. Every person who has had an acute appendicitis, whether he had been operated upon or not, requires analytical work in the bowel. Practically every acute and subacute appendicitis is secondary to an infection in the bowel which the removal of the appendix does not cure. Of course, if after these operations the patient is totally symptom free and remains so in a general sense, no more attention perhaps need be given. But the vast majority of them do not remain symptom free in a complete sense, and thus it is best that these examinations be made either before or after such operations.

One needs but to hear the constantly unfolding similar story of patients thus operated upon to note the accuracy of this statement, and it would be worth while for our surgical friends to sit in my own or some other physician's office to learn this simple fact. When at operations on the appendix (or the gall-bladder) adhesions of the non-embryonic type are

met with, this case should be judged as one of intestinal toxemia and sent for study, and, if necessary, treatment.

Ptoxis, Gastric Dilatation, Dilatations of Sections of the Bowel, and Incompetence of the Ileocecal Valve, Etc.

—Ptoxis is mostly a congenital condition, excepting those of degrees which follow pregnancy. It commonly is accompanied by intestinal toxemia and when this is secondary to the ptoxis (using this term in a general sense) the handling of the ptoxis is all that is required. But not uncommonly a primary intestinal toxemia is co-existent, and if the ptoxis is treated successfully, in a few months or a year or more all of the symptoms have nevertheless returned.

This is the main reason for the non-substantial results in medical treatment of ptoxis. To leave a primary intestinal toxemia under a ptoxis is meeting the situation only half way, and practically in its poorest half. There have been several hundred cases of people who have been put through the “bed-diet-rest” treatment for ptoxis by well-qualified men with excellent results at the time, which were sustained for a short period, and then had a reversion back to the original condition, but whose health was finally restored when their intestinal toxemia had been properly taken care of. Every instance of ptoxis requires a careful biologic study of the intestine, and if a primary intestinal toxemia exists this must receive attention.

Other than pyloric stenosis, habitual over-eating and ptoxis, there is no greater cause of gastric dilatation than chronic intestinal toxemia. Intestinal toxemia is likewise the cause of dilatations of the cecum and ascending colon, and incompetency of the ileocecal valve. When these are mentioned in the history of patients, and confirmed on examination, the bowel should be carefully examined for biological errors and treated accordingly.

More than half of the instances of intestinal toxemia show one or more of the dilatations. The conditions come from the baneful effects of intestinal toxemia on the neuromuscular apparatus of these hollow structures and involvements of the sympathetic ganglia and nerves which supply these structures,

the same type of degeneration as was mentioned in connection with constipation and colonic stasis.

Diseases and Conditions Classified in Other Specialty Groups.—In the lists presented from this on throughout this chapter, and especially in the last part of this volume, many disorders, diseases and conditions will be enumerated. These in the author's experience were met with as present at the time or were recorded in past histories of patients. Only those that were actually encountered in definite ways in the five thousand cases are presented. Up to this a few general symptoms and gastro-enterologic conditions have been mentioned. A large proportion of the cases were sent to me by a specialist in other disorders for handling of some digestive condition. When an intestinal toxemia existed that could be benefited, often to his and my own surprise, the condition being treated by the specialist disappeared or became markedly improved. This experience has repeated itself so many times and was so definite and striking that many diseases and disorders can be offered as a part of the intestinal toxemia subject. These will be presented more in detail in connection with the section on clinical cases and the results of treatment. At this time, the fact simply will be mentioned only to serve as a guide, and if they are present in the past history or are present at the time, a biologic study of the intestinal canal is always warranted.

The reader is not to understand that in each of them an intestinal condition is always and ever the etiological factor. It is to such a large extent, however, and to such a surprising degree, that one often wonders at the large amount of work and degrees of interest that are being carried out among these specialists in the way of investigations in which the intestinal canal is absolutely neglected as a cause.

However, there is another side to this matter, and one quite as important clinically, and that is that an intestinal toxemia, while in an instance apparently not a distinct etiological factor, is not infrequently a most important contributory one. Time and time again this was a proven fact, and time and time again the benefits brought about in the intestinal condition paved the way or helped materially for the specialist

to bring about a worth-while result, a result which before that was not accomplishable nor could be sustained. This to me is a very important matter and one that no specialist can ignore.

It also is not always possible to say that an intestinal toxemia in this or that condition is etiological totally or only contributory and to what extent. The human being is a biologic animal with various reactions and results from the same disease and conditions. Even inoculation to identical infections themselves will cause disease in one and not in another. When it causes a disease, some die quickly, some die much later, with all degrees between, and some recover. This is true of every infectious disease known, and intestinal toxemia is an infection process. So it is true with an identical infection or disease in different people, and experimental animals, that a running play of pathologies in kinds and extents ensues, and also that different accompaniments and aftermaths are seen. One needs but to think a moment of pneumonia, typhoid, diphtheria, rabies and practically all infectious diseases to acknowledge that the above statements are self-evident.

There are in all infectious disorders factors of susceptibility which influence inoculation and course, factors we know practically nothing about and which are generally not estimable. Death takes place sometimes to our surprise, and recovery the same way. It, therefore, is not always deducible when a disease or disorder exists, how much a different etiology operates as a cause for the condition then present. And likewise in such a condition, how much an intestinal toxemia is distinctly etiological and how much contributory. But clinical work has proven that intestinal toxemia is a most important factor, weaving throughout many of the diseases and conditions of all the specialties, and a chronic case should be studied along these ways to do full justice to the patient in most of the little understood conditions.

Age Incidence and Standing of Symptoms.—The ages of these patients when seen by me are recorded as follows :

10 to 20 years	2 per cent.
20 to 30 "	17 " "
30 to 40 "	21 " "
40 to 50 "	24 " "
50 to 60 "	25 " "
Above 60 "	11 " "

It is here important to remember that in the young no worth-while discerning ability of minor states of ill health exists, this usually not being present until some years after adolescence. Then, too, the symptoms when present are usually transitory in the child with quick recoveries and but little attention is paid to them. It is not until the individual comes well into the adult years that observational and discerning ability of symptoms becomes keen enough for their record on the patient's mind and judgment.

In this block of cases the length of time that states of ill health had existed, or that enough suggestive symptoms were present, were compiled. These in lengths of standing were:

Existent since childhood	2 per cent.
35 years standing	3 " "
30 years standing	5 " "
20 years standing, or more	6 " "
15 years standing, or more	9 " "
10 years standing, or more	13 " "
5 years standing, or more	14 " "
3 years standing, or more	21 " "
1 year standing, or more	27 " "

From the above it is evident that an intestinal toxemia can start in childhood, and even though there be no symptoms recording back to the early years, the condition commonly does start at an early age and generally is disregarded. In these, many times, efforts and attempts had been made at every sort of diagnosis but the right one, and, largely according to the personal equation or the type of medical attendant, quite an array of beliefs, diagnoses, treatments and even operations are recorded by the patient, in the straight run of histories studied. Those who had had one abdominal operation to correct the symptoms complained of were 10 per cent., those who had had two operations were 7 per cent., and those who had had three or more were slightly short of 1 per cent. The surgeons should

remember that a person may have an intestinal toxemia for years with little or no local symptoms in the abdomen and then have them develop and perhaps continue. In such instances unless the symptoms pivot around some organ in definitely suggested ways (such as a gall-bladder, stomach or duodenum, appendix, etc.) operations would be of no benefit excepting perhaps in a moral way or because of the rest in bed. For the uncomplicated case of intestinal toxemia operations are not curative and commonly make patients worse, and also they cannot be diagnosed by an x-ray examination or even a visual examination with the abdomen opened.

Leaving out definite pathologies existent in the cases before seen by me or noted on physical examination, and recording only the symptoms the patient presented of what may be termed strictly functional types, the following briefly represents the percentages noted in this group of histories. In another 5000 group they might be different in way of additional symptoms or percentages. These are sufficient, however, to give some idea of the symptoms present in the strict primary toxemias, and the list gives a fair idea of their frequencies. In them many detailed types of symptoms are generalized in one group.

Intestinal flatus (definite states)	75	per cent.
Fatigue	70	" "
Constipation	64	" "
Regurgitation (belching)	42	" "
Neurasthenic symptoms	42	" "
Mucus from bowel	41	" "
Stomach weight and pressures	41	" "
Abdominal distress, pains and tenderness	41	" "
Anorexia and fitful appetite	34	" "
Stomach hyperesthesia	32	" "
Irritability	27	" "
Diarrhea	26	" "
Hypotension	25	" "
Stomach hyperacidity	23	" "
Loss of weight	22	" "
Indigestion (digestive disturbances)	22	" "
Headaches	20	" "
Mental depressions	19	" "
Anemia	18	" "
Abdominal distensions	17	" "

Nausea attacks	16	per cent.
Joint pains and swellings	15	" "
Stomach pains and distress	15	" "
Insomnia (fitful sleeping)	14	" "
Vertigo (dizziness)	13	" "
Debility, physical	12	" "
Cardiac palpitation	12	" "
Vomiting	11	" "
Neurotic manifestations	11	" "
Subject to colds	9	" "
Abdominal colic or cramps	7	" "
Head pains	7	" "
Chest pains	7	" "
Arm pains	7	" "
Corporeal pains	7	" "
Asthmatic attacks	7	" "
Hypertension	7	" "
Vasomotor phenomena	7	" "
Skin disorders	6	" "
Obesity	6	" "
Functional eye symptoms	6	" "
Spinal pains and tenderness	6	" "
Intolerance to starch, sugar and fats	6	" "
Hiccoughs	5	" "
Drowsiness, post-prandial	5	" "
Lumbago	5	" "
Jaundice attacks	4	" "
Leg pains	4	" "
Back pains	4	" "
Dyspneic attacks	4	" "
Chills and chilly sensations	3	" "
Unconsciousness (attacks of)	3	" "
Melancholia	3	" "
Head noises	3	" "
Intolerance to acid foods	3	" "
Halitosis	3	" "
Epileptic attacks	2	" "
Deafness	2	" "
Pyrexia attacks	2	" "
Bulimia	2	" "
Corporeal itching	1	" "
Rectal pains	1	" "

To the above, of course, could be added subjective and objective symptoms found in the various disorders met with which are consequent to or in which the toxemia is definitely contributory. For the completion of symptoms present or found, the reader is referred to the physical examination and

to the clinical section of this volume. The above merely records the character and location of symptoms stated by the patient at the time of history taking or noted on examination.

LONGEVITY.

It is universally accepted that man has a span of life fixed at the "three score and ten," and it has been preached for years in practically all text-books in medicine that time is the total factor in accomplishing age and decay. There are now enough proven facts to suggest that there are far more potent factors in the things that happen in the course of life of the individual. Some of these are bound up in heredity, hygiene, climate, toxins from infectious diseases, food deficiencies and excesses, physical trauma, physical apathy, psychic trauma and apathy, and endocrine cycles.

The recent lengthening of the span of life of the human being (about 37 years) has largely been accomplished by the saving of young lives, but the most important influences are undoubtedly bound up in Dr. Charles H. Mayo's statement that 85 per cent. of deaths are due to some form of infection. It is the author's opinion that these unknown "forms of organisms menacing the human races yet to be discovered" are almost totally those of the intestine bound up in the production of intestinal infective and toxemic conditions. Until this side of the question of life is paid more attention to by medical men and biologists, no really worth-while advance in longevity will be accomplished.

CHAPTER IV.

THE PHYSICAL EXAMINATION OF THE PATIENT.

Not much of other than suggestive interest is elicited on physical examination. Like the history, in which the grouping of symptoms is more or less vague, existing or extending to any part of the body and rarely pivotal in diagnostic ways, the objective findings on physical examination are rarely definite, and yet they are often quite suggestive of the condition. The reader is referred to the clinical section of this volume for more data regarding the various diseases.

PROCEDURES IN PHYSICAL EXAMINATION.

Hair.—Beginning at the head, dry crackly hair is often seen in the toxemias. When the hair up to a period of life had been what may be termed normal and since then continuing in the dry condition, especially when the period started about the time of the beginning of other symptoms, these impart an interest. Of course there are local conditions of the hair and scalp in which dry hair occurs and so far as this sign is concerned these conditions would have to be thought of and eliminated.

Eyes and Eyelids.—An eyelid symptom of interest is chronic blepharitis when trachoma can be eliminated. Chronic red lids with more or less blocking of the Meibomian glands at the lid edge is commonly a symptom of focal infection located elsewhere than the eye. Of like interest are chronic congestive states of the conjunctiva both of the interior of the lids and on the eyeball. When it is seen on the eyeball the congestive state fades out toward the cornea—the lids, therefore, having to be drawn down to observe its presence. When present, the conjunctiva often appears thickened, as if there were a catarrhal process in it. In fact in most instances a catarrhal process does exist (catarrhal ophthalmia), this commonly being an extension of a hypertrophic rhinitis.

Other symptoms of interest are opacities in the vitreous and various degrees of foggiess noted when looking at the fundi. "Dancing spots" in the vision and scintillating scotomatous attacks usually have no objective findings in the eye, yet these are present in intestinal toxemia cases. Sluggishness of pupillary contractions to light, with irregularities, should excite interest although most times the pupillary reflex is normal.

Among other ocular disturbances often found in these intestinal conditions, which should be thought of in this connection are eczema of the eyelids, arcus senilis symptomatica, pale conjunctival membrane (anemia), chronic tarsitis, anemia of the retina, spasm of the retinal arteries, simple retinitis, sclerosis of the retinal vessels (arteriosclerosis), simple or serous retinitis, and retinitis pigmentosa. Recurrent attacks of iritis (toxic) and bilateral exophthalmus with hyperthyroidism are always suggestive, as is amblyopia and temporary amaurosis otherwise not accounted for. To these may also be added corneal ulcers, keratitis and about one-fourth of the instances of refractive errors.

Ear, Nose and Throat Conditions.—No more definite conditions are due to chronic intestinal states than those classified as simple chronic and the chronic hyperplastic (hypertrophic) rhinitis. In simple chronic and hypertrophic rhinitis (the most common of chronic nasal conditions), the first may be somewhat due to obstructions as the second causes them in turbinate hypertrophies. But neither the causative in the first or the resulting in the second are as important matters of interest in the treatments conducted by the nose and throat specialists for these conditions, as it is that they should have intestinal handling instead of only local washes, oils, operations, etc.

General medical terms are used in an etiologic way in the text-books on these conditions, like "gouty," "rheumatic," "diabetic" and "strumous," these being passed along from one author to another. Of course "diabetic" means something in clinical medicine, but "gouty," "rheumatic" and "strumous" are hopeless and only cover a depth of ignorance. I am willing to accept that atrophic rhinitis (ozena) is probably a strictly

local disease of the nasal mucous membrane, but both the simple and hypertrophic forms of chronic rhinitis are always due to intestinal toxemias. One has only to observe the nasal mucosa of these cases when housed up in warm rooms in the winter, and lacking fresh air, sunshine, exercise and fresh fruits and vegetables (need of which increases intestinal toxemia of any form) to prove this. At least a thousand of these nasal cases have been studied as to seasons of the year, and what happens to these cases when the intestinal toxemia is cleared away? Without any nasal treatments they practically all have gotten well with only the simplest local handling of the nasal mucosa. What generally is termed "nasopharyngeal catarrh" and "chronic post-nasal catarrh" belong distinctly to these intestinal states as the main and important contributory causes.

Another condition in which much benefit has been derived is sinusitis which after operations and various procedures has failed to clear. A chronic intestinal toxemia may not only be a primary etiologic factor but it is also a contributory cause of significance enough to make recovery impossible. Sinus infections of any or all of the head sinuses are, when chronic, symptoms sufficient for intestinal study and treatment. Among my cases there have been about a dozen of chronic sneezing lasting years of time, all having made recovery or marked improvement on treating the intestinal condition. Chronic trouble with "head noises" and attacks of functional deafness have also been cleared up by intestinal handling. The commonest cause of permanent deafness, namely otosclerosis, which is today believed to be hereditary, should be studied from the intestinal standpoint. I have reason to believe that its cause is more intestinal than transmitted, and children who show the development of this condition should have an intestinal biologic study and handling before the condition is well established.

Oral Conditions.—Of interest here in so far as the tongue is concerned is its pallor (as well as the mucous membrane of the whole mouth) in anemia, and the large soft, flabby and serrated type. Occasionally it is more red at the tip and perhaps the edges a little way back, and small blisters (sometimes painful) may be present. Attacks of congestive states of the

tongue and especially under the tongue should suggest an intestinal significance.

Mouth.—A symptom noted while the tongue is being examined is the odor of the breath. When local conditions in the mouth and tonsils can be excluded, a foul breath should always excite interest. Practically all of the instances of this that are chronic, or more or less present at times, are due to toxic states in the small intestine, the resorbed sulphate products coming out in the expired air. A "sour" breath also should call the possibility of the condition to mind.

The gums should always be examined for general swelling and sponginess. Ulceration along the line of the gums as well as "canker" sores of frequent recurrence are symptoms worthy of note.

As has been mentioned elsewhere, apex abscesses of teeth are more often due to intestinal organisms carried by the blood stream from chronic infected and bowel states than from the mouth itself. When bad dentistry can be excluded, dead and abscessed teeth should not be considered as totally a local affair. Every such case deserves study of the intestinal biology and treatment where errors in this region are found. Where these have been or are now present, dead or abscessed teeth should not be considered a sign of small or only local significance.

Mention is here made for the first time of one of the most classic symptoms of chronic intestinal toxemias in the mouth, one not always present and only possible of eliciting when the upper molar teeth are present. When examining the hard palate the gum margins of the upper molars should be observed. If they edge the teeth with a pronounced red line (generally very narrow) and there is a pronounced white line (also very narrow) on the palatal side to this, and then the membrane extends in appearance like the rest of that covering the hard palate, an intestinal toxemia is present in practically all of the instances. This finding is the outstanding single classic symptom of the mouth. The intestinal tract should also be studied in congestive tongue states and lymphatic pharyngitis.

Tonsils.—The tonsils should always be looked at for enlargements with faucial congestions and, even when the tonsils appear normal, should have one and, better, two methods of examination which are more thorough than the practice of only observation as is done today. Firstly, pressure of the finger should be made directly backward on the anterior pillar onto the tonsils and note made of what is expressed on the tonsil surface from the crypts. A thin seropurulent discharge is the most significant, and a cheesy material from the crypts is next. Secondly, swabs should be made from the supratonsillar fossæ, hard-surface media inoculated, and the bacteriology studied. Any streptococcus or staphylococcus is always significant. Thirdly, if swabs are not used, a fairly accurate bacteriological diagnosis can be made by the appearance of the anterior pillars of the fauces. In streptococcus infections a narrow red line will run along the free margin, while in the staphylococcus infections the red margin is wide, perhaps spreading across the width of the pillar. In mixed infections these characteristics might not be so sharply outlined. All the local infections of the mouth and nose need attention in two directions, namely, the local and the intestinal.

Nails.—Nails will often show the presence of anemia, and those which have prominent transverse grooves, when a recent illness can be eliminated, seem more likely to be instances of intestinal toxemia. I have sometimes thought that innumerable white spots in the nails had some significance but have never been sure. The hard, brittle and longitudinally striated nails are not uncommonly seen.

Blood-vessels.—The next step would be to observe whether the brachials are visible above the bend of the elbow at the inner side. When they are both visibly pulsating and distinctly palpable, confirmation of an arteriosclerosis diagnosis may be made by a similar examination of the femorals and the use of an ophthalmoscope in noting the condition of the retinal vessels. Arteriosclerosis is a frequent accompanying and resulting state from a long-standing neglected intestinal toxemia.

As a rule the most common finding in blood-pressure is that of a hypotension, this being represented in both the systolic and diastolic pressure, but more particularly in the first. It is not uncommon, however, to see a marked status of intestinal toxemia in a normal or a heightened pressure. As the result of a long-standing intestinal condition (especially those grouped in the saccharo-butyric toxemias) which has been neglected with resulting disease of the vessels, it is not uncommon that various degrees of change in the myocardium (chronic myocarditis) and perhaps in the kidneys (usually interstitial sclerotic changes) can be diagnosed. In addition the intestinal canal should be studied in hyperglycemia, hypercholesterinemia, uricemia, simple and pernicious anemia, low-grade bacilleмии and so-called "acidosis."

Habitus and General Condition.—In persons of the asthenic habitus (long thin type) the rule is for the intestinal toxemia to be of the putrefactive or mixed types, and very largely the intestinal toxemia is of the secondary sort and, therefore, of no significance in the primary treatment. However, one cannot always be sure of this because primary types of toxemia are often found in these people and examination should be conducted to prove or disprove this.

In the sthenic individual (short thick type) the toxemia is usually of the fermentation and acid type (more particularly belonging to the general classification of saccharo-butyric toxemias), these being more particularly conditions in which the symptoms are abdominal in location, with or without abnormal metabolic associations.

What has been stated in connection with habitus very largely runs clinically true with the general condition of the body. In the asthenic type the general body condition would be poor, the tone low, perhaps anemia will be present, while in the sthenic type the subcutaneous fat tissue would be ample, and the individual would perhaps be quite obese. Many of the so-called constitutional types of obesity are those of obesity as a symptom of chronic intestinal toxemia in which the toxemia has inhibited proper oxidation of the body. The same is true of hyperglycemia and hypercholesteremic states, which

are largely resulting conditions from some fermentative type of intestinal toxemia.

Heart Conditions.—While it is generally agreed that distinct lesions of the valves of the heart are due to rheumatism, syphilis or some definite type of an infection, there are not a few instances of lesions of the heart valves which in all probability have come from an infection that has gained entrance into the general system by way of the portal circulation and through the liver. In a general way, however, where a distinct lesion of the valve or valves exists, one is inclined to the conclusion that there is only a modified significance in any direct suggestive way as to the presence of an intestinal toxemia. This, however, is not true in the production of less distinct or functional types of heart murmurs, because these have been found often enough in connection with intestinal toxemia to prove an association between the two.

In those who are middle-aged and older various degrees of enlargement of the heart are not uncommonly found. Usually this is uniform throughout the entire organ, the compensation being satisfactory and apparently always remaining so. Following along in significance are the instances of chronic myocarditis not infrequently seen even in those who are still young.

Of much interest in this connection are states pertaining to the rate of the heart. While it is most probable that distinct bradycardia (slow heart) is not associated with intestinal toxemia, tachycardia almost always is due to it, and this is true of tachycardia connected with hyperthyroidism. Various alterations in the heart rhythm can certainly be due to an intestinal toxemia. Among these are sinus arrhythmia, paroxysmal tachycardia and extrasystoles. While diseases of the arteries and island degenerations in the auricles with interference in impulse conduction into the ventricles may be explained on the basis of pathology, the reader is asked to remember that any and all of these pathologies may come from long-standing and neglected intestinal toxic states.

Lung Conditions.—Diseases of the lungs are quite well known at the present time, although there are certain pulmonary conditions that are still not understood. These are

largely those of the so-called allergic conditions of the bronchial mucous membrane, the asthmas, especially those of the so-called essential type, and more or less generalized fibrosis non-tubercular in origin.

While chronic infections of the lungs may explain the occurrence of some of these, there have been such marked improvements in patients who have had these distressing conditions in the chest that the author feels that intestinal toxemia (while perhaps not directly etiologic) is a strong contributory factor in not a few of the chronic lung conditions, especially in streptococcic and staphylococcic infections. The intestinal canal should always be studied in chronic bronchitis, the so-called essential asthma, and the allergic conditions.

Abdominal Conditions.—The conditions most often seen in primary intestinal toxemia are those connected with dilations of the stomach, the right colon, and perhaps the entire colon, it being often possible to diagnose them on physical examination.

The next most common type of case is the so-called atonic one with low intra-abdominal pressure, the organs being relatively ptosed according to maximum limit figured from the bony structure, the width, depth and length of the abdomen. Not a few of these are instances of intestinal toxemia which have come on in a contributing or secondary sense to the ptosis, gastric, ileocolonic, and colonic in nature (secondary toxemias). The possibility of a primary toxemia should not be lost sight of here.

The next common are conditions in which the liver is distinctly hardened, usually throughout its entire structure but more particularly in the left lobe. Not uncommonly there are varying types of cirrhosis, these not going on to the lethal states as in true portal sclerosis, but cases in which at operation the liver is found to be distinctly hob-nailed. There is little doubt in my mind that these more partial forms of cirrhosis of the liver are due to intestinal toxemia, and but rarely directly to the effects of drinking of spirituous or malt liquors. Of interest here, too, would be instances of tenderness in the median fissure of the liver (gall-bladder region),

these being most commonly cases of chronic cholecystitis, with or without stones, which condition is almost always secondary to a chronic intestinal toxemia.

Not infrequently there exists more or less tenderness in various portions of the abdomen, usually in the region of the appendix or the ascending or descending colon, with perhaps the gut wall more palpable over the right colon than the left, these always being signs of significant interest. The intestinal canal should be studied in gastric hyperacidity, achylia, gastrectasis, ileal and colonic stasis, peritoneal adhesions, neurosis, ptosis, dilated cecum, appendicitis, ulcerative and simple types of colitis, spastic and atonic colon, intermittent diarrhea, sigmoiditis, proctitis, intestinal indigestion, chronic cholecystitis, cholangitis and cholelithiasis, hepatic cirrhosis, "bilious" attacks and chronic constipation.

Cutaneous Symptoms.—The color of the skin would be significant in an anemia, while that of a distinct light-brownish tinge, noticed best in the face, over the sternum, in the flexures of the elbow, and posterior knees (popliteal region), is quite characteristic of distinct putrefactive conditions. Of course one should always watch for mild states of jaundice, and whether the condition of the skin is normal, or distinctly dry and tending to be scaly. The latter condition with dry hair is a frequent combination of intestinal toxemia.

A careful search should be made for any of the various skin eruptions in which intestinal toxemia may be etiologic or one of the main contributing factors it is necessary to correct to bring about distinct benefit. Most important also are the dermatographic reactions, especially the so-called Sergeant's white line indicative of suprarenal deficiency or an expression of vagotonia from breakdown of the gland defenses from the long-standing intestinal toxemia. Also of value are the hyperthyroid reactions, these being secondary to the toxemias.

Intestinal biologic study should be made in recurrent urticaria, angioneurotic edema, toxic dermatitis, erythemias, chronic eczemas, lichen planus, pruritus, and purpura simplex rheumatica.

Joints.—The joints should be examined especially in connection with so-called gouty conditions and the various forms of chronic arthritis. Tenderness, deformity, grating, cracking, creaking and various other symptoms of joint conditions may or may not be signs of significance. Intestinal study is required in osteitis deformans, spinal osteoarthritis and gout.

Nervous System.—Note should be made of any disturbance of the muscles which may reveal an increase or exaggeration from normal motility. Various types of tremors, especially those of the hands of the fibrillary type, are more or less eluding. Perhaps the most characteristic symptoms of those that may be elicited on physical examination are the groups which may go under the term sensory hyperesthesia. These may be elicited in the chest, the lower abdomen, in the back along the spine, or on the scalp and in fact most anywhere throughout the body.

In individuals who are distinctly of the neurasthenic or hysterical type, disturbed superficial cutaneous and the deep reflexes of the upper extremities and that of the exaggeration of the patella (unless other causes can be gotten to explain them) should excite interest in the possibility that an intestinal toxemia may be causing them. Biologic study of the intestines is required in neurasthenia (fatigue neurosis), recurring neuralgia, neuritis (brachial and sciatic), sensory polyneuritis, recurring headaches, hyperesthesia, psychasthenia, emotional depressions, chronic backaches, emotional instabilities, organic diseases of the nervous system and mental states when other causes are not plainly suggested or can be reasonably assumed.

Endocrine States.—Since glandular imbalance and dysfunctions when not congenital occur from the environmental factors of which the intestine is the most important of all, and in fact is a most common cause, the following are always to be studied as of intestinal origin, namely, exophthalmic goiter, hyperthyroidism, and mild states of hypothyroidism, as well as hypo-adrenalism. Added to these have been not a few instances of striking results in cases of mild types of diabetes, and the so-called constitutional types of obesity.

Kidneys and Bladder.—Infections and chronic disease of these organs are commonly of intestinal origin. Among them notably are pus kidney, kidney and bladder stones, chronic cystitis, nephritis and albuminuria. In these the biology of the intestines should be studied.

Rectal Examination.—As a rule, the physical examination is finished with an examination of the anus and rectum, especially the rectal mucosa. The anus has significance in a neurotic and nervous type of individual whose nervous state may have been produced by a long-standing intestinal toxemia, and the appearance of the rectal mucosa has significance in a differential diagnosis more particularly between atrophic and hypertrophic states of mucous membrane change.

This may be taken, not only as diagnostic of intestinal toxemia, but also the possibility of a resulting pathology which might help to elucidate the question as to whether the intestinal toxemia is primary or secondary to some other condition.

Examination of Children.—Since many intestinal toxemias start in childhood, the instances of diarrheic and constipated conditions should have intestinal study. To them should be added backward physical development, fitful, peevish and irritable states, intestinal indigestion, mucus conditions, and so-called "acidosis."

SUMMARY.

In addition to the findings above suggested, when the following occur in the past history of the patient or are present at the time of the examination, they should be judged as presumptive evidence of the presence of a chronic intestinal toxemia:

DISEASES AND DISORDERS OF INTERNAL MEDICINE.

Acidosis.	General debility (vital and physical).
Albuminuria.	Hypertension.
Anemias.	Hyperthyroidism.
Angioneurotic edema.	Hypotension.
Arteriosclerosis.	Hypothyroidism.
Asthma.	Myalgias.
Diabetes mellitus.	Myocarditis.
Endocarditis.	Nephritis, acute and chronic.

DISEASES AND DISORDERS OF INTERNAL MEDICINE (*Continued*).

Obesity.	Rheumatism.
Pancreatitis.	Septicemia.
Peritonitis.	Thyroiditis.
Pneumonitis.	Vasomotor disturbances.

DISEASES AND DISORDERS OF THE DIGESTIVE SYSTEM.

Abdominal adhesions.	Hyperacidity, hyperchlorhydria.
Achylia gastrica.	Ileocecal incompetency.
Appendicitis.	Indicanuria.
Atonic colon.	Intestinal stasis.
Colitis.	Neurotic abdomen.
Colitis, ulcerative.	Omentitis.
Constipation, chronic.	Proctitis.
Diverticulosis and diverticulitis.	Pruritus (anal).
Erosions of the stomach.	Pyloric obstruction.
Gall-bladder infections.	Pylorospasm.
Gastric disturbances.	Redundant colon.
Gastritis, chronic.	Spastic anal sphincter.
Gastro-enteroptosis.	Spastic colon (constipation).
Gastro-intestinal atrophy.	Spastic recto-sigmoid apparatus.
Hemorrhoids.	Ulcer (gastric and duodenal).

DISEASES AND DISORDERS OF THE HEART.

Angina pectoris.	Myocarditis.
Coronary sclerosis.	Nervous heart.
Endocarditis.	The cardiac irregularities.
Functional heart conditions.	T-wave negativity.
Hypertensive heart disease.	

DISEASES AND DISORDERS OF THE NERVOUS SYSTEM.

Anxiety states.	Migraine (hemicrania).
Depressions.	Neuralgia.
Epilepsy.	Neurasthenia.
Functional mental symptoms.	Neuritis.
Hysteria.	Organic neurology.
Manic-depressive psychoses.	Psychasthenia.
Menopausal neuroses.	

DISEASES AND DISORDERS OF THE EAR, NOSE AND LARYNX.

Deafness.	Rhinitis; acute colds in the head;
Hay fever.	chronic rhinitis.
Labyrinthine disturbances; vertigo	Sinus disease, chronic and recurrent.
(intestinal).	Tonsillitis, acute, chronic, and
Laryngitis, chronic.	purulent.
Nasopharyngeal conditions.	Tracheitis, chronic.
Purulent chronic middle ear disease.	

DISEASES AND DISORDERS OF THE EYES.

Amblyopias.	Ocular manifestations of nervous diseases.
Blepharitis.	Optic neuritis.
Cataract.	Postoperative infections.
Choroiditis and choroidoretinitis.	Retinal diseases (retinitis).
Conjunctivitis.	Scotoma (central, false, toxic and scintillating).
Episcleritis.	Uveitis.
Errors of refraction.	Vitreous conditions.
Iritis.	
Keratitis.	
Neuroretinitis.	

DISEASES AND DISORDERS OF THE SKIN.

Acne.	Nails.
Acne rosacea.	Pemphigus.
Dermatitis.	Pityriasis rosea.
Dermatitis herpetiformis.	Pruritus.
Eczema.	Psoriasis.
Erythema exudativum multiforme.	Purpuras, the.
Herpes zoster.	Scleroderma.
Lichen ruber planus.	Seborrhea.
Molluscum contagiosum.	Urticaria.

DISEASES AND DISORDERS OF JOINTS AND MUSCLES.

Arthritis, atrophic and hypertrophic arthritis, osteo-arthritis, and spinal arthritis.	Myositis.
	Postural defects.
Arthritis, chronic (atrophic, hypertrophic, osteo-arthritis, rheumatoid, deforming). Spinal arthritis.	Various joint conditions.

DISEASES AND DISORDERS OF THE URINARY SYSTEM.

Acute suppuration of the kidney.	Pyonephrosis.
Calculus, renal and ureteral.	Renal tuberculosis.
Cystitis.	Ureteritis and ureter stricture.
Perinephritis.	Urethritis.
Prostatitis.	Vesiculitis.
Pyelonephritis, chronic.	

DISEASES AND DISORDERS OF THE FEMALE ORGANS.

Cervicitis, endocervicitis and metritis.	Pelvic adhesions.
Cystitis (pyelitis).	Postoperative ileus.
Oöphoritis infections.	Pruritus vulvæ.
Parametritis and pelvic cellulitis.	Puerperal sepsis.
Pelvic abscess.	Salpingitis.

DISEASES AND DISORDERS OF CHILDREN.

Acidosis and alkalosis.	Gastritis, acute and chronic.
Acrodynia.	Intestinal colic.
Allergies.	Malnutrition.
Anorexia.	Rheumatism.
Celiac disease.	Sinuses.
Dilatation of the stomach.	Tonsils.
Gastralgia.	Vomiting.

For a more complete consideration of the above-mentioned diseases and conditions the reader is referred to the clinical section of this volume found from Chapter XIV to the end of the book.

Depending upon the nature of the findings and their importance (to which the findings on examination of the office urine are added), the attendant decides whether or not to carry the investigation further. In a well-established condition existent for years in which pathology is deemed permanent and irreparable it may be decided to carry the investigation no further. This rather hopeless attitude against the carrying out of further investigation commonly presents itself.

There are, for instance, certain organic types of heart disease of advanced forms, marked ulcerative colitis in terminal degrees of systemic states, stones in the kidney, urinary bladder, gall-bladder or pancreatic ducts, well-established organic disease of the central nervous system, advanced arteriosclerosis, diabetes mellitus, etc., in which the desirable attitude to take would be to handle the case in the quickest way to get results, namely, either medically or surgically. Unless there is time and it seems or becomes justifiable, well-established pathology should be handled immediately, because the rule is that when such advanced definite disease in organs exists nothing worth while can be accomplished by biological study or treatments. In such instances one is not given the time or encouragement to carry out the investigations, which may take weeks of time, and commonly are not appreciated by patients, and sometimes are directly discouraged by the family physician in charge.

In these instances one may make mention of the importance of an intestinal state an etiological or contributing factor, but

only as an assumption which could not be proven one way or the other without laboratory investigation.

However, if the findings or the suggestions or assumptions on the case warrant it, if the time factor is easy, and the conditions, understandings and facilities for investigative work are present, one may proceed with the necessary further examinations. Before this is undertaken, of course, any x-ray, laboratory or special apparatus examinations should be cleared away. In starting out for a biologic diagnosis everything necessary for the complete examination of the patient should have been performed and valued, recorded, decided definitely upon, or left to one side for the time being.

Not infrequently more or less simple types of treatment, such as rest cures or office procedures, can be conducted while the investigations are being carried out. In this way some degree of benefit may be accomplished, and the more ignorant type of patient kept satisfied for the time being by something being done. One begins then by placing the patient on the standard test diet mentioned, the foods being carefully weighed and the specimens collected properly, and the examination started on his arrival.

As a rule the specimen for the blood chemistry can be taken on the morning of the fourth or fifth day (before breakfast, of course). It commonly is necessary to collect more than one warm specimen of feces (sometimes two or three), in which instance the dieting is continued, or the carbohydrate or the protein test diets are swung into while more specimens are secured. The latter two diets are 72-hour test-dieting plans, and the biologic series have to be marked as per plan of diet from which they were secured. It is a general rule to allow no medications by mouth during the test-dieting periods. In a few words, investigations are started to make a biological diagnosis of an intestinal infection or toxemia.

CHAPTER V.

THE EXAMINATION OF FECES.

BACTERIAL DIAGNOSIS.

MacNeal, Latzer and Kerr Method.—Various attempts have been made to estimate and standardize bacteria in feces. Among these may be mentioned the one of MacNeal, Latzer and Kerr, in which the numbers and quantity of bacteria, nitrogen determination and differential count are utilized. After mixing with a spatula or mortar and pestle, in order to obtain uniform test samples, a 1:100 suspension of feces in saline solution is prepared in a 500 c.c. glass-stoppered flask as a basis for study. The total quantity of bacteria is determined by three different procedures: the Eberle-Klein (plain slide) microscopic method; the method of Winterberg (Thoma-Zeiss blood-counting chamber); and the gravimetric method of Strassburger (fractional separation by washing and centrifuging, and weighing of dried bacterial residue).

The nitrogen in the bacterial residues is determined by the Kjeldahl method. The average daily bacterial nitrogen in the feces and the percentage of total fecal nitrogen are calculated. Differential bacterial counts are made by a modified Gram-staining method in which the decolorizing is done with anilin-xylol followed with methyl alcohol or the usual Gram differential method of staining.

Rettger Method.—The following has been suggested by Rettger as a complete routine method and serves to fair purpose if a standard diet is initially part of the procedure: (1) Microscopic examination and differential counts of Gram-stained smears of feces; (2) the use of the fermentation-tube method of Herter and Kendall, in which glucose, lactose and saccharose are employed; (3) cultivation on Loeffler's blood serum and in gelatin stab culture; (4) plate counts on sugar-free agar aerobically, and on lactose agar anaerobically; (5) the use of Endo plates; (6) determination of spore-forming

bacteria in lactose agar plates and in milk tubes, and (7) employment of acetic acid glucose broth tubes for aciduric organisms. My experience with this method is that it has a very limited value in diagnosis. The technical and most important of fecal examinations as routined by the author is described in a later chapter.

Method of Tsuchiya.—Another method which was offered because of its quickness for clinical purposes is that of Tsuchiya¹, which was not intended for a detailed bacteriologic study of all the bacteria present in the feces, but of a few bacterial groups whose dominance is directly responsible for the character of the flora.

On a general classification of some groups, a cultural and microscopic technic is presented. This technic was run one hundred times in conjunction with my own methods, and the results were so erroneous, conflicting and, in my opinion, so valueless, that this method is not advised. There is no simple and easy way in this work, and unless a complete and careful examination is made it would be best that the work be not undertaken at all.

Lieb-Chapman Method.—Another method of bacterial diagnosis was devised by Lieb and Chapman. This is a combined one of the determination of virulence (toxigenicity) as devised by Falk in estimating *B. diphtheriæ* and known as electrophoresis, and complement fixation studies with group antigens. They used a modified technic in isolating and identifying the *Escherichia coli*, the *Proteus aërogenes* and other Gram-negative bacilli, after Levine's eosin methylene blue media and blood agar, a special agar-sodium phosphate with bromthymol blue as an indicator, and undertook to study toxicity by the electric method of speeds against standard strains.

They use as standards of the Falk method, non-toxic strains when above 60, and toxic ones below 60, 45 being very toxic. On this part of their examination they state as follows: "We found that we were able, in most instances, to distinguish a normal from an abnormal stool by the presence or absence

¹ Tsuchiya: Arch. Inter. Med., 36, No. 5, p. 636.

of the toxic strains of *B. coli*. The exceptions were generally infections by streptococci and *C. welchii*." These exceptions were that "in the former instance the stool is usually poor in bacteria due to inhibitions of *B. coli* by streptococcus toxin. In the latter we usually observe a marked increase in *C. welchii* but the strains of *B. coli* have normal toxicity indices." In the antigen studies they grouped up in the following ways, having found the single antigen method too erroneous:

Name	No. of strains in antigen.	Name.	No. of strains in antigen.
<i>Aërobacter aërogenes</i>	5	Other anaërobes	8
Typical non-hemolytic coli ..	5	<i>Streptococcus viridans</i>	7
Atypical non-hemolytic coli ..	5	Non-hemolytic streptococci ..	3
Narrow hemolytic coli	6	Hemolytic streptococci	5
Wide hemolytic coli	6	Non-hemolytic enterococci ..	4
<i>Salmonella</i> group	6	<i>Enterococcus viridans</i>	4
<i>B. typhosus</i>	1	Hemolytic enterococci	4
Dysentery group	9	<i>Staphylococcus albus</i>	4
<i>B. pyocyaneus</i>	2	<i>Staphylococcus aureus</i>	4
<i>Proteus</i>	4	<i>Gonococcus</i>	7
<i>Alcaligenes</i>	4	<i>Neisseria</i> (meningococci and	
<i>Friedlander</i>	3	catarrhalis group)	5
<i>Diphtheroids</i>	3	<i>Monilia</i>	3
<i>C. welchii</i>	7	<i>Hemophilus</i> (influenza, etc.) ..	2

Quite candidly they admit the errors in complement fixation work in this connection in the following statement: "We found that by the usual methods of complement fixation, a given bacterial antigen might give a strongly positive result with a given blood, but if other bacterial antigens were also employed, they would also give positive results." This they state "is due to the fact that all bloods contain varying amounts of a substance which will give positive results with any bacterial antigen."

Their report in conclusion is as follows: "Electrophoresis and bacterial complement fixation have been applied to a study of the intestinal flora. It is possible by these methods to differentiate a normal from an abnormal stool. The exact type of bacteria and, generally, the actual abnormal strain can be readily identified. A combination of the two tests is very highly recommended in preference to either one alone. The

correlation and agreement between the two tests are remarkably close."

Feeling that the above method was worthy of study, fifty instances of definitely toxic and infective individuals were taken as a standard of study with fifty normal controls. These had been examined by the author's method, the infections straight and in association being known. The results of the studies proved that the electrophoretic method of Falk was of such limited value in intestinal diagnosis, positive and negative findings being mixed up in such erroneous ways, that its use was discontinued even in association with the complement fixation method.

In definite infections, non-toxic figures and toxic ones were met with so often in the controls, that as a means of definite diagnosis its limitations in values rendered it practically useless. One must ever keep in mind that there are far more bacterial infections in the intestines than strains of *Escherichia coli*, the streptococci and the *Clostridium welchii*, and any method of diagnosis which cardinalizes on these or a few more bacteria, is bound to be in error in at least 90 per cent. of the instances.

Elsewhere in this volume matter pertaining to the limitation of complement fixation work in intestinal diagnosis is presented. Since many of the infective organisms are endogenous, and therefore non-toxin producers, and the majority of intestinal toxemias are associated infections, it must be manifest that complement fixation work with single antigens against strain infections, or group antigens against definite strains of bacteria, is bound to fall short of worth-while practicability. No means of antigen tests against blood serum will ever be of value.

Therefore, my deductions from the methods of diagnosis of Lieb and Chapman are that they represent another instance of interesting effort but worthless application, and even were they of some value in a diagnostic way, they are worthless for biologic therapeutic suggestions.

TEST DIETS IN FECES EXAMINATIONS.

The first essential of feces examinations is that they be made only on stools which represent a known diet. The examination of casual stools, as is the practice today, is not only worthless but most liable to lead into error. The causes of errors are use of the casual stool specimens representing a bacteriology and chemistry greatly influenced by the foods eaten during the days previous, and different foods can and do change the bacteriology and chemistry of the bowel contents for long enough periods of time to enter most confusing states and factors.

We have, in addition to these factors, several more which we cannot control, such as the effect of constipation, stasis, the activities and amounts of fresh air, etc., the patient had lived under in the days previous. But the food intake can be controlled and this is most essential for a proper examination of feces. This point was originally brought out by Praussnitz¹, and elaborated by Ledden-Hulsebosch², Strassburger³, and Schmidt⁴, the latter two of whom devised diets for the purpose. The work of these observers had to do with the examination of the food detritus of stools and fermentation tests and had nothing to do with the bacteriology in essential ways. The latter is really the most important of all.

It makes but little difference what the standard of diet is as long as it comprises the necessary amounts of proteins, carbohydrates and fats properly interspersed in intervals during the day and of sufficient but not too ample caloric value and not containing too much wood fiber (cellulose). The "general" and the "detailed" test diets of Strassburger and Schmidt may be employed but these have not been as practical in my own work as the one mentioned below. Their use, however, is in no sense deprecated.

Of course, before this examination is started, a gastric test meal, preferably by the fractional method is made, perhaps also

¹ Praussnitz: *Zeitschr. f. Biolog.*, 35, p. 335, 1897.

² Van Ledden-Hulsebosch: Berlin. Julius Springer, 1899.

³ Strassburger: *Deutsch. Arch. f. klin. Med.*, 61, p. 571, 1898.

⁴ Schmidt: *Berl. klin. Wochenschr.*, Nr. 41, 1898.

a blood chemistry, the dermal food tests when suggested, and a roentgenographic examination performed before the individual is placed on the author's test diet (modified in children down to seven years of age according to the weight). Often, so as not to load up the intestinal canal and feces with barium (which may take days in being eliminated entirely), the roentgenographic examination follows the collection of stool and urine specimens for biologic studies.

AUTHOR'S DIET AND RULES FOR OBTAINING SPECIMENS FOR TESTING
THE DIGESTIVE FUNCTIONS OF THE INTESTINES.

Attention should be paid that the diet and rules are followed precisely. The reasons for this are that careful and extensive chemical and bacteriological examinations are made from the specimens obtained and sent, and unless the diet and rules are followed accurately the laboratory findings may be misleading.

When you start with the diet you are to follow it for 72 hours (or 3 full days) without interruption. The stools and urine voided during the first 48 hours (or 2 full days) are not to be saved. Beginning at the 48th hour from the start of the diet, void urine and start with an empty urinary bladder. All the urine that is voided from the 48th to the 72d hour (voiding for the last time at the 72d hour) is saved and thrown together to estimate and record the exact amount voided; only 8 ounces are to be saved and sent to my laboratory. A portion of the stool passed during this time (from the 48th to the 72d hour) is also saved and put in some kind of a container, preferably a wide mouthed glass bottle such as a small vaseline bottle or a small mason jar. If the bowels do not move employ a small enema of warm tap water to obtain the specimen. Put your name on the specimen containers and promptly send them to my laboratory. Take no medication of any kind when on this diet. If you are distressed with constipation, take a low enema of tap water.

Diet to be Followed.

Morning—Oatmeal gruel, made of about 30 grams or 1 ounce of oatmeal, 10 grams or $\frac{1}{3}$ ounce of butter, 120 grams or 4 ounces of milk and 200 grams or 6 $\frac{2}{3}$ ounces of water (all strained). In addition to the gruel take 1 $\frac{1}{2}$ thin slices of well baked bread with butter liberally applied, and one egg cooked in any form.

11 A.M.—Milk, 8 ounces or a glassful.

Noon—A good sized piece of steak or roast beef, chopped or cut into very fine pieces (about 80 grams or 2 $\frac{2}{3}$ ounces) and served on a slice of toast; one bowl (about 180 grams or 6 ounces) of mashed potatoes with 10 grams (or $\frac{1}{2}$ ounce) of butter.

4 P.M.—Milk, 8 ounces or a glassful.

Night—Same diet as at breakfast.

Water may be taken as desired.

When the specimens are ready bring them to the office the next morning before breakfast, and ask to see the nurse in charge for further instructions.

The last sentence of the above slip is most essential. At the time the specimens are left at the laboratory (the patient still being on the diet) one of several procedures is conducted, this being for the purpose of obtaining a warm fresh specimen from as high a level in the bowel as possible for the bacteriological examination. To this end a "high" enema using the final wash water may be employed, although biologic failures and errors are liable to happen by this method.

My usual procedure is to pass a duodenal tube and, when the tip is *in situ*, to flush the intestines from above with warm tap water (104° F.), or a hypertonic solution, keeping the patient about until one obtains the second or preferably the third bowel ejection. The latter represents the contents from the ileum and right colon and with proper cultural methods represents a viability of obtained organisms much higher in percentages than that of the cold stool specimen the patient had brought in. Sometimes the patient is kept on the diet for an extra day or two and purged with salines or castor oil (only the second, third or fourth ejection being employed).

Because the biologic studies must be started not later than thirty minutes after the warm stool is ejected (and it must be kept warm until it is) these bowel movements must occur in close association with the laboratory, the patient kept on the diet until such is obtained. Occasionally one may obtain the proper type of specimen after several movements have occurred by passing a proctoscope and draining the rectal contents. The technical examination of these warm specimens will be described later on.

Special Types of Diets.—The above type of diet does not answer in every instance for the technical studies, it alone answering in about one-half of all instances. In addition, special types of diets to stimulate the activities of certain types of organisms are commonly in order. The first of these is a high carbohydrate test diet in which an array of sugars are present, this being for the purpose of generally stimulating

in the small intestine all the organisms of and those biologically associated with the *Escherichia coli* grouping (as well as many others Gram-negative). The diet then prescribed is totally that of starches, sugars, vegetables and fruits, in which at each meal during the day are taken: dextrose 2 grams, lactose 2 grams, dulcitol 1 gram, mannitol 1 gram, and raffinose 1 gram.

The procedure in collecting the specimens is then identical with the one given above, and that represented in the high protein diet about to be mentioned. One of the most important phases of the biologic study is the estimation of anaerobes. Some of these are sufficiently stimulated by the regular test diet, others by the high carbohydrate diet, and others require a high protein stimulation. When the protein test diet is used toward the latter end of the procedure, a diet is arranged comprising large amounts of beef and mutton, fish of any kind, eggs, at least two in the day, cheese, milk, gelatine foods taken thrice daily, oatmeal, legumes, to which are added for practicability, bread, butter, orange juice, chocolate, cocoa and water. One, therefore, often uses three types of stimulating diets, each at different times, each collected in warm specimens with studies from each started at once, and each run as separate procedures all the way through the biologic work, which for a proper diagnosis of laboratory time takes from one to twelve weeks on each.

METHODS OF EXAMINING THE COLD FECES SPECIMEN.

The examination of the cold specimen of feces answers several important purposes. It gives an idea of the digestive powers of the entire digestive tract, serves for chemical as well as fermentation tests, and particularly is of value for chemical examination of admixture substances. Its greatest importance in this connection, however, is for it to serve as a further basis for test diet selections and therapeutic dieting. Its value in the biologic examination is limited to such information as may be gathered in bacteria counting of the Gram differential stain slide.

Macroscopic Examination.—The macroscopic examination forms an important part of the whole procedure, and this alone may serve to suggest what the subsequent diets should be. The odor of the normal stool may be described as mildly excremental. When pathologic conditions exist it may have a distinctly sour odor from butyric, acetic and various other fermentation acids, or it may be distinctly malodorous from putrefaction.

The normal color, a seal brown, may through a mixture of blood become distinctly blackened. The clay-colored stool found in certain states of intestinal toxemia, from reduction of the bile coloring matter by bacteria, as well as in obstructive states of the biliary ducts, is quite characteristic. One may also notice the fatty or glistening stool, occasionally seen in instances of late chronic pancreatitis, in states which cause a rapid transit through the small intestine and the right half of the colon, and occasionally in perfectly normal people.

Of significance would be the appearance of gross flakes of mucus, blood, pus, parasites, etc. Surface mucus usually is added to the feces in the lower end of the large intestine, and while this may be of significance in itself it does not possess the interest of mucus which is incorporated with the food detritus, the latter appearing under the microscope in the form that is well known. As a rule, the consistency of the normal stool is sufficient in firmness to be cylindrically shaped on one end and somewhat tapering on the other.

The stool of certain forms of atonic constipation is usually large in size or presents itself in small scybala, one ball packed against the other. This is the characteristic stool of the putrefaction type of toxemia. On the other hand, the stool of the saccharo-butyric type presents itself as soft, mushy or fluid, usually yellowish or yellowish-green in color, and, if mushy or fluid, presents innumerable bubbles of gas.

One then proceeds to a closer examination by taking up a small amount of stool with a wooden spatula and mixing in a mortar and pestle with a small amount of water to about the consistency of thin catsup. A portion of this is taken, mixed with more water and the reaction judged on the basis

of the *pH* value. The *pH* of the normal stool is about 7, that of the acid type being lower. The best apparatus to use for this is the LaMotte Duplex Indicator.

The next step is to transfer a small part of the specimen in the mortar to a microscopic slide, spread it rather thin and examine it under the microscope for various pathologic findings. Of first interest, of course, would be the noting of the ova of parasites, amebas (seen easiest in the warm specimen), etc. Sometimes this is quite confusing because the vegetable cells of not a few of the ordinarily eaten substances closely simulate the ova of parasites. Familiarity with stool examinations soon teaches one to detect the difference between the two which is generally made easy by shifting the low-power ocular to one of medium amplitude or employing the de Rivas method of separating out the ova.

With a very low-powered microscope or a strong magnifying glass remnants of connective tissue and tendons of chopped meats may be readily examined. They are usually of whitish-yellow color, thread-like in appearance and solid in consistency. Where large quantities of tendon fibers are met with one's interest in some form of gastric disturbance should be aroused. The muscle tissues appear chiefly as small brown-colored rods which can be easily broken up by pressure and show muscular structure under the microscope. Where these occur in large quantities an intestinal cause should be suspected.

Occasionally connective tissue and muscle occur combined as meat remains. Where there is doubt whether one deals with tendonous tissue or rolls of mucus the treatment with acetic acid of a thread under the microscope causes the thread-like structure to disappear, whereas in mucus it appears more distinctly, or at least does not disappear.

Potato remains appear like small sago-grain, glassy transparent granules, which may easily be mistaken for flakes of mucus. Such substances are treated with any of the iodine preparations (Lugol's solution), color blue with starch, purple or red with etherodextrine, or show no change with acrodextrine. Fat remains may be noted by the clay-like color

and consistency, or occurring in the form of small, soft, light-yellow lumps (calcium soap).

By this simple macroscopic examination, best conducted with the specimen crushed between two microscopic slides and a magnifying glass, mucus is readily observed. Larger shreds or strips are easily recognized as glassy transparent flakes, occasionally colored yellow by bilirubin, with irregular ragged outline. It may also be seen as hyaline mucus islands and yellow mucus granules, although such may be simply particles of starch substance. Examination of the flakes under the microscope usually clears any doubt that may be present.

Microscopic Examination.—The next step is to transfer a small portion of the stool from the mortar to a microscope slide, usually with the addition of a drop or two of water on the slide, the specimen being spread thin and examined at once. Schmidt recommends that three such slides be made. The first, the specimen as it comes from the mortar, pressed on the microscope glass with a cover-glass. The second is thoroughly mixed with a small drop, 36 per cent. (official) acetic acid and heated for a moment over a flame until it begins to boil and then covered with the cover-glass. The third is rubbed up well with a small drop of a strong solution of iodo-potassium iodide (iodine, 1.0; potassium iodide, 2.0; aq. dest., 50.0).

The first slide is used for a more detailed examination of the granules, globules, etc. Usually in this, isolated fragments of muscle fibers occur, clumps of the yellow salts of calcium, colorless soaps, isolated potato cells and various other substances such as the chaffy remains from the oatmeal gruel, remnants of cocoa (nibs).

The second specimen, in which the fat has been melted temporarily, gives an idea of the entire fat content, this appearing in small flakes of fatty acid scattered over the entire preparation. In this, the larger salts of calcium and soap flakes usually disappear.

In the third preparation the potato remains ultimately assume a slight violet shade, not blue, although most of the starch substance is colorless or stained a yellow from the

iodine. Where there is distinct limitation in starch digestion, the granules may appear mahogany in color.

One particularly searches in the microscopic examination of the food detritus for fragments of muscle in large numbers, needles of fatty acid and soap, drops of neutral fat, numerous groups of potato cells, as well as parasite eggs, pus, blood and substances of like kind, and especially definite quantities of neutral calcium phosphates or ammonium magnesium phosphate crystals, both of which latter, when in distinct amounts, are indicative of an enteritis usually in the small intestine and often accompanied by a distinct toxemia.

CHEMICAL EXAMINATION.

The next step in the examination may be described as the chemical one. Of course in this connection would come the pH value already described, and strips of the red and blue litmus paper can be employed or a drop of the feces may be added to a weak solution of neutral azolitmin in a test-tube.

For a number of years I have used the sublimate test suggested by Schmidt, in which a small quantity of feces (to which more water has been added) is placed in a flat vessel (preferably one of the bottoms or tops of Petri dishes) and treated with a concentrated watery solution of bichloride of mercury, stirring and allowing to stand until the next day. The normal feces are generally colored red from hydrobilirubin, and the particles colored green show the presence of unchanged bile pigment (bilirubin). On occasions the entire content of the dish may be green.

My experience with this test, however, has been that it is not of much clinical value, and should be discontinued since the advent of the van den Bergh or the icterus index test.

FERMENTATION TESTS.

The Schmidt Test.—Schmidt, who has done more than anyone in advancing the so-called fermentation test, describes his test as follows: From the fresh excrement (which has been stirred up, but not yet thinned with water) a small amount (it should be about 5 grams; the estimated amount, however,

is sufficient for our purposes), about the size of a walnut, is taken with one of the wooden spatulas and put into the lower vessel of Strassburger's fermentation tube (in hard stools less is taken; in soft more; in fluid stools the lower vessel is filled entirely). In this vessel it is stirred up with water with the aid of a wooden spatula, and then the rubber stopper is put on, care being taken to exclude air bubbles. The rubber stopper is now taken from the little tube "b" and the tube filled with tap-water. This is then closed by turning over the vessel "a"

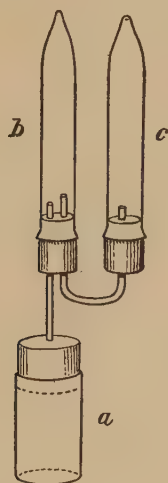


Fig. 1.—Schmidt's Fermentation Set.

together with the small stoppers, and the vessel "c" (empty) connected with it; "c" has an opening on top and acts as an ascension-pipe (Steigrohr).

Note should be taken of the figure indicating how far the connecting glass tubes enter into the lumen of the three vessels. When the apparatus is put together, it is placed for twenty-four hours in an incubator and heated to 37° C. If gas is developed from the feces within this time, this collects in "a" or "b" and a corresponding amount of water is driven into the ascension-pipe "c." The height of the water in the ascension-pipe is noted, the vessel "a" is opened, and the reaction of it is tested with litmus paper, and compared with the reaction

before the test was instituted. There exists a modification of the fermentation tube by Dr. Baurmeister (Braunschweig) which has the advantage of being handier and stronger.

Normally no gas, or little, is formed in the incubator-test and the original reaction of the feces undergoes no important change. If gas is developed to such an amount that one-half or more of the ascension-pipe is filled with water, then pathologic conditions are suggested, and if at the same time the reaction has become distinctly more acid, carbohydrate fermentation (the positive result of the fermentation test) has occurred; if it has become distinctly more alkaline, albumin putrefaction has occurred.

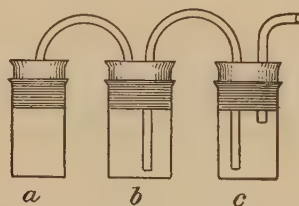


Fig. 2.—Baurmeister Fermentation Set.

In the former case, the opened glass "a" gives off the odor of butyric acid, in the latter case it gives off a very decided putrefactive odor. The color of fermenting feces is generally brighter, that of putrefying feces is generally darker. The suspicion of pathologic fecal fermentation can often be confirmed by the mere inspection of the original feces, namely, if they appear light brown and foamy, react acid, and smell of butyric acid.

In rare cases the fermenting and the putrefying processes so interact upon each other that, notwithstanding the copious formation of gas, the reaction and the other characteristics (color, odor) there will be no change of significance.

Author's Modification of Schmidt Test.—My own experience with the Schmidt fermentation test has not been satisfactory, although I employed it at least a thousand times. The element of error is considerable, and so definitely is this true, that I have modified the test in ways that are more satisfac-

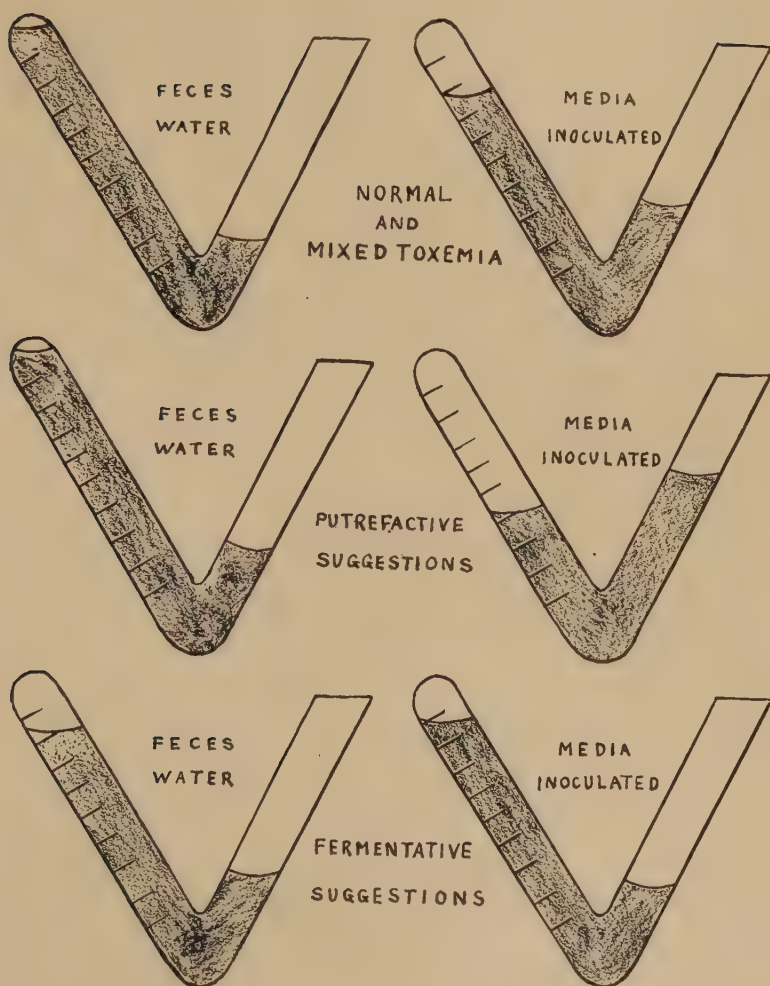


Fig. 3.—Different Degrees of Gas Results in Feces and Water and Media Inoculated Fermentation Tubes the Author Employs.

This method cannot be depended upon for diagnosis and is mostly of service to suggest types of diets. There are too many opposite and conflicting results for them to be depended upon for bacteriological or even chemical conclusions. For example, the commonest and most difficult basic type of toxemia to diagnose and control, namely the mixed form, in at least half of the instances gives gas results corresponding to normal (the same irregularities are seen in the other basic types) so that here the test may not even be of value for dieting.

tory and conclusive, although, even with my own test, errors commonly occur. Without going into the subject of bacteriology in connection with this form of testing feces (for which it is almost useless), the Schmidt test gave marked variations in gas results in distinctive conditions, and realizing that the gas result was solely and entirely one of bacterial activity, I have modified this test to that which follows and which I would recommend in place of the above procedures.

Occasionally it is possible by a double procedure to check one test against the other. While the results are not always distinctive and should not be depended upon alone, here and there it has been possible to acquire laboratory data of some value by a cleaner and more easily performed method, and one which gives a better gas index in a bacteriologic way than those already mentioned.

For this purpose V-shaped fermentation tubes are used, the right limb having the same capacity as the left. The capacity of the left limb from the top to the crotch at the bottom is 15 c.c., and the tube is divided off in tenths, each space between the markings having a capacity of 1.5 c.c. Two of such tubes are used in testing each specimen of feces. In one the mixture of feces and water is exactly as in the procedure recommended by Schmidt.

For my purpose we use 5 c.c. of dry hard feces, 7 or 8 c.c. of the stool when it is mushy, and 10 to 15 c.c. of stool when it is fluid, this being proportioned between these amounts according to the consistency of the original specimen. This amount of feces is mixed in a mortar to which tap-water is added sufficient in quantity for the whole to fill the left limb of the tube to slightly beyond the crotch on the right side. The distance of the contents of the tube on the right limb should not be more than 2 cm. from the crotch.

In the second tube is placed a 2 per cent. dextrose-bouillon which is then inoculated with one or two wire loops of stool. This is thoroughly mixed in the media, both tubes are placed in the incubator and the gas results read in twenty-four hours. In about half of the instances one can notice a disparity in the gas results in the two tubes.

Schmidt states in his fermentation test that "Normally no gas, or little, is formed in the incubator-test." This I have not found to be always true. Not uncommonly as much as 10 or 20 per cent. is noted. As a rule, in my way of testing, when a saccharo-butyric type of fermentation is present the content of gas in the media tube would be distinctly less or just equal to the content of gas in the water feces tube. And, on the other hand, if a status of putrefaction predominates, the content of gas in the media tube would be distinctly higher than that in the water and feces tube.

Even in normal stools the content of gas in the media tube would be as high as 20 per cent. Also of interest in this connection in examining stools of the same normal individual is the difference in gas results on different types of diet. Those on a strict meat diet usually do not run much more than 10 per cent., while those on a strict vegetable diet usually exceed 30 per cent. The media tube in the saccharo-butyric type of toxemia usually does not show much more than 10 per cent., while those in the indolic forms often exceed 60 per cent.

I wish here to refresh the reader's mind to the fact that while these fermentation tests may here and there serve to some clinical purpose, they are quite as liable to lead to error. However, they should be performed, because occasionally they may be a link in the chain of laboratory diagnosis. There are so many types of bacterial combinations in which fermentation and putrefaction are associated, and in which one is consequent upon the other, that while these fermentation methods may be sufficient for diagnosis with some, as a rule they are quite worthless.

EXAMINATION OF THE GRAM DIFFERENTIAL STAINED SLIDE.

We now consider the next step in the laboratory examination of the cold specimen, and that is the examination of the Gram differential stained slide, the counting of bacteria, arriving at a percentage result between the Gram-negative and Gram-positive, and an appreciation of the bacterial picture as it presents itself to the eye. In normal individuals on the standard

test-diet, differential count between the Gram-negative and Gram-positive is about 70 Gram-negative to 30 Gram-positive. This is not an arbitrary standard because it is different in normal individuals and different in the same individual at different times, even when on the same diet.

However, it serves for a purpose because when the stool is predominantly Gram-negative or Gram-positive, suggestions as to the type of toxemia that exists may be gleaned and more especially suggestions as to the type of diet to be employed for a short time with the patient. In the putrefactive or indolic states usually the Gram-negatives are high in percentages, whereas in the saccharo-butyric, and more particularly the fermentation states, the Gram-positives are increased beyond the percentage of 30 and may even be the dominant bacteria in the field.

Bacteria Counting.—For the purpose of obtaining a differential count between the two staining types or organisms, various methods have been devised, the most common one being the spreading of a thin stool specimen on some one of the many types of blood-counting slides and counting the bacteria within the lines as one does white or red blood cells.

The usual blood-counting chamber has a very small area on the glass on which bacteria can be spread, and thus gives a very poor chance of accomplishing a worth-while result. The feces specimen on the glass should cover a considerable area so that different sections of a specimen may be counted. For this purpose there is no better means than to employ an ordinary microscope slide. The specimen spread upon the slide should be thin and as free from food detritus as possible. Just the smallest quantity of stool, preferably such as would adhere to an inoculating wire, should be applied to two or three drops of water on a slide, spread over a considerable area (covering about a quarter of the entire surface), be as free from particles of feces as possible, and when dried on the slide represent just a visible film on the surface. Any of the methods of Gram differential staining may be used.

For the purpose of visualizing small areas, small enough for the bacteria to be counted, the author suggests the use of

discs fitted in the ocular eye-piece, these in form being according to the observer's selection. The ones employed in my work are diagrammed as follows, the visual field occurring in areas always in focus when the high-powered objective is employed:



Fig. 4.—Author's Types of Inserts in the Ocular Eyepiece of the Microscope for Counting Bacteria.

It will be noted that the openings are arranged so that the field or fields are in focus without manipulating the adjustment. These dials can easily be made out of thin tin.

One counts several dozens of such areas from different sections of the slide and notes the results in percentages.

Having had sufficient experience in the appreciation of bacteria as viewed through the microscope, certain types of organisms in the cold specimen of feces would be readily appreciated. While those of the *Escherichia coli* groups are more or less different in their morphology as they occur in this type of specimen of stool, they are seen in several forms

and are more or less readily identifiable. The *Clostridium welchii*, of which the capsule may be identifiable here and there, can be counted, usually occurring in the normal specimen about eight to the field. When they are above or below this it usually has significance.

The short, narrow Gram-positive *Clostridium putrificus* is usually identifiable, and when these occur in any perceptible numbers, they have significance. There are usually a few spirochetes of innocent type in stools. When these are met with in distinct numbers they have significance because they may represent types of spirochete infections not heretofore described, infections capable of producing distinct toxic results and which are identifiable only in this way.

Beyond these points, the various cocci, diplococci, streptococci, staphylococci are to be noted and charted. The Gram-positive single and double cocci are denizens of the normal stool, but when they occur in large numbers they have distinct significance. No Gram-positive staphylococci or streptococci should be present in normal stools. One must distinctly observe, too, the presence of free spores in the stool, and whether they are Gram-negative or Gram-positive. Most of the *Clostridium* genus, almost all of which are inimicable in the intestinal canal, contain or send off Gram-positive spores. One must also note the presence of large stick-like organisms, both of the Gram-positive and Gram-negative types, these occurring in many different shapes and sizes and most of which are saprophytic in nature. These have significance because the saprophytes as groups comprise some of the most important of intestinal infections, and are in themselves capable of producing most distinct clinical conditions.

Beyond these few points of the simple study of the Gram differential stained slide nothing more need be stated, and it must now be apparent to the reader that, while up to this time certain data has been accumulated, this data still does not represent a biologic study of any real scientific clinical or therapeutic value so far as the biology of the intestinal canal is concerned. But if up to this point certain facts and findings here and there, be they ever so insignificant, have been gained

(or often if none have been gained), and the case clinically is definitely suggestive of a toxemia, one cannot stop at this point.

The only conclusions thus far possible by the examination are usually negative as to suggesting definite infection types of organisms. We often, though, gain findings of seeming significance, but about all that usually can be concluded is that we have gained certain suggestions that would be of value in a dietetic way, and that in a biologic way we are still completely "at sea."

The author now wishes to present the type of record slip he employs for the ordinary examination of cold feces. (See following page.)

EXAMINATION OF THE WARM FECES SPECIMEN.

Where warm specimens have been obtained on the different types of testing diet, Gram differential stained slides and percentage countings should also be made of them at the time of starting the culture procedures.

The examination of the warm purged specimens begins as soon as they are obtained. Whether the specimens are secured from standard, carbohydrate or protein test diets, the examination, especially the bacteriological, must be begun within thirty minutes from the time the stool is voided. Intestinal organisms are viable but a short time after they have left the intestine. Separate smears on microscope slides are prepared and these may be examined later as already explained. In this examination the pH values are again estimated as with the cold specimens, and the general character observed, the fermentation tubes (feces and water, and media) are again set up, and perhaps the food detritus and abnormal findings observed through the microscope as was mentioned before.

In addition to these, Gram differential slides are again made and examined, the general character of the flora observed, the identification and numbers of the different bacteria and spores carefully looked into, and all possible suggestions and facts pertaining to the symbiotic groupings and associations with differential countings carefully observed. It is in these latter matters that experience is the most valuable helpmate,

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FECES, COLD SPECIMEN.

[illegible]

and experience cannot be taught by words but only from work, memory and recording of past errors. From a beginning when all is dubious and practically nothing could be observed by the procedures mentioned thus far, the addition of this simple microscopical examination of the Gram-stained specimens, after a few years of work, becomes more understandable the more it is observed, and it does occasionally happen that one needs to go no further than this stage for the diagnosis (and not infrequently the true biologic suggestions) of which further work will prove the accuracy.

However definite suggestions these may be, the examination should not stop here but be carried through all of the necessary extra procedures, because it is the rule that the findings thus far will prove to be erroneous, in which instance the whole biological work would be fundamentally on a wrong basis and consequently valueless, both in diagnosis and as therapy. It is often of help in what has been offered (as well as generally throughout further studies) to use the micrometer. While the same organisms differ in size (and in many of their other morphological characteristics) there is a generality in them all in a microscopic field that could be taken advantage of for identification. This is true whether examining organisms from stools or from media because a prominently seen organism matching up by micrometer often has its significance. For this purpose the measurements of the most prominent of these organisms are given later, with their descriptions.

Inoculating the Hard Media.—One now starts the real work of diagnosis and the gaining of biologic therapeutic suggestions. For this purpose a normal saline suspension (very thin) of the warm specimen is used largely as the inoculating material, the whole procedures (normal saline solution and all media, even the hard) being kept at blood heat temperature (98.5° to 104° F.). The first step is to inoculate the hard media. For the first of these (Koch procedure) a peptone salt solution is also made, but in the streaking the saline only is used. The procedures are as follows:

Plating by the Koch method using each one of the following, gelatin or agar, infusion agar, blood agar after preliminary emulsification in broth or peptone solution. Two sets are made, three are grown aërobically, and three anaërobically (see below).

Streaking Petri plates of agar, blood agar or solidified blood serum. Two aërobic and two anaërobic (see below).

The rapid transfer method (almost entirely studied with milk) has not helped materially although it may be important to study the lactic acid and other bacilli and the *Clostridium welchii*.

Inoculation of Fluid Media.—The inoculation of the fluid media is now in order, and for this purpose both the aërobic, anaërobic, and a combination are employed. For the anaërobic, solid media studies are made with the media in tubes rather than in Petri dishes, because in both solid and fluid media, a large series of tubes of the one medium (each to be taken out from time to time) are employed, and these when stoppered permit the opening of the containing jars and the re-establishment of the anaërobiosis for further study (usually 7 tubes of each medium are employed).

If the pyrogallic acid-caustic soda or potash method is employed, saturation of the plugs with the mixture of acid and alkali and the tubes hermetically sealed is called for. The anaërobic method employed by me is that devised by McIntosh and Fildes as modified by Smillie. The aër-anaërobic method is well enough accomplished by fermentation tubes (closed limb) or the media in extra long and narrow test-tubes, and the use of freshly boiled media, quickly cooled, and covered with mineral oil after inoculation. The media usually inoculated by me in the original set-up are represented in the following. According to the suggestions gained in examinations thus far, occasionally but rarely, a modification of the number is engaged in. This depends upon the skill and the experience of the examiner and it takes years to learn this one small modification. So that the work might be as accurate as possible, the routine should be the entire set-up.

Hiss calcium carbonate broth as modified by Bolduan.	Levulose.
Lactose litmus agar.	Maltose.
Litmus milk.	Dulcitol.
Two per cent. dextrose bouillon.	Saccharose.
Meat extract gelatin.	Insulin.
Meat extract agar.	Isodulcitol.
MacConkey's bile acid agar.	Inositol.
Veillon's agar.	Adonitol.
Robertson's cooked meat, Tinsler's modification.	Arabinose.
Coagulated egg white broth.	Amygdalin.
Robertson's alkaline egg, Kahn's modification.	Salicin.
Kahn's inspissated ox serum.	Sorbitol.
Potato.	Raffinose.
Hiss serum water.	Dextrin.
Conradi-Drigalski, Park & Williams modification.	Erythrite.
Endo-medium, Kendall's modification.	Glycerin.
Conradi's picric acid, green medium.	Galactose.
Levine's eosin-methylene blue medium.	Mannitol.
Park & Williams tetanus toxin broth.	Loeffler's blood serum.
Avery's oleate agar or acid broth.	Rosenow's brain broth.
Teague & Travis cholera medium.	Park & Williams mallein broth.
Simmon's medium.	Eulow's sugar-free medium.
Burbank streptococcus method.	Ellermann's medium for <i>B. fusiformis</i> .
Lactose.	Dunham's solution.
Glucose.	Media for vibrio.
	Alkaline Dunham's solution.
	Gelatin stab.
	Agar broth.
	Media for spirochetes.
	Agar ascitic medium.

(Other data regarding media are found in connection with the different organisms.)

The original set-up is approximately 110 tubes for anaërobic and about 35 for aërobic. These are added to as the studies proceed, usually about 200 tubes of media being employed for the one specimen of stool. Where the three types of test diets are examined, their numbers are multiplied throughout. In some instances as many as a thousand tubes of media are necessary for complete study of the single case.

Morphology.—Motility, shape and size of bacteria are studied by the hanging-drop method, and development through several generations, and to study spore formation, etc., the Hill method is employed. The spores are stained with the

Kendall or Hiss, the capsules by the Welch method, the polar bodies by Neisser and the flagella by Pittsfield. In the differential staining the Gram method serves of best purpose, although the acid fast of Ziehl-Neelsen, the Fränkel-Gabbet or Wright's are occasionally called for to help in identification.

Cultures.—The isolation of pure cultures of aërobes is by the plate and streak methods, the Barber method occasionally being essential; these are done at any stage of the biologic studies. For the anaërobes both the plate and fluid media are employed, these also being done at any stage of the biologic studies. The carbohydrate media are used for reinoculation and studies of reaction and gas formation (see gas ratios of Smith for different aërobes and cultural characteristics). The streptococci isolation may be accomplished by the Burbank method or by direct plating on dextrose agar or dextrose broth inoculation.

Members of the alcaligenes, dysentery and other groups are separated in part by endo-media (as well as typhoid and paratyphoid groups identified and separated from organisms of the so-called mucosus capsulatus group and the others by the multiple methods suggested). Spirochetes are best observed in the Gram-stain specimen of stools and perhaps identified by special media. The pallidum does not occur in stools but the others mentioned do; these may be isolated on agar ascitic medium anaërobically. Occasionally rabbits, guinea-pigs and white mice are necessary for obtaining pure cultures and to study virulence (see works on bacteriology).

The staphylococci group are best studied by plating or streaking on agar or gelatin; the streptococci on agar or dextrose agar plates; the pneumococci by mice inoculation; the alcaligenes agar, gelatin and milk; the dysentery organisms by the sugars; typhoid, paratyphoid, coli, cloacæ and the proteus group need not be specially presented here (see works on bacteriology and cultural characteristics). The same pertains to the lactic and aciduric organisms, anthrax, pyocyaneus, and the many others.

For identification and separation the different sugars are very important. Inoculation of Von Drigalski and Conradi

media is also used to study the typhoid and the paratyphoid. The Veillon method is employed to study the anaërobes and accomplish their isolation. Cantu's method is occasionally employed for the study of the proteus vulgaris. Manifestly the many facts pertaining to general and special bacteriological matters, cultural characteristics, stains, etc., need not be repeated in this volume. This part of the work belongs properly to volumes on bacteriology, of which there are many excellent ones. The main points, however, are presented in the chapter on morphological and cultural characteristics.

Overcoming Difficulties Encountered.—There remain, however, two matters important to present. When running through the various media and growing methods there are not a few difficulties to be met with. One of the most important of these is that organisms may be present which will take on none of the media and methods. There are two ways to handle this question: The first is to stimulate the bacteria within the intestinal canal by carbohydrate or protein test dieting, in that way accomplishing more viable forms for the original inoculations, and the second is to use a feces extract of the patient's stools, portions of which are added to the fluid and sometimes the solid media. As experience multiplied, the initial food stimulation method has become more and more common until today it has become almost routine with the author.

To this end when the warm specimens from the standard test diet are secured the patient is immediately swung onto the total protein or the total carbohydrate diet, in most instances specimens obtained from both being employed. In this it makes but little difference what the sequence is, but the sequence of standard test diet, protein and carbohydrate is the rule most often followed. The patient's total time on test dieting then would be a matter of from 9 to 11 days, the latter figure allowing interval time for the shifts to be made between meals, trips to the laboratory, etc.

Whatever then would be the laboratory set-ups employed on the standard test diet warm specimen would be duplicated with the protein and carbohydrate test diet specimens also.

This, of course, triplicates the work and adds another ten days of time to the time required, namely from 2 to 12 weeks as the instances for proper biological studies would be called for in the case. There is no way to shorten this excepting by the use of two set-ups for the standard test diet specimen, one for the different media as they are made up and the second the same media in the second set-up, to each of which fecal extract of the patient is added. To this end some of the warm specimen of feces is mixed with a small quantity of sterile normal saline solution, quickly centrifuged to throw out the food detritus, the supernatant portion passed through a Berkefeld filter and the obtained fluid added to the different media in the second set-up in lots of about 1 c.c. in each.

Since lytic substances are often present even in the purged specimens, these may defeat the very purpose one tries to accomplish by this means; oftentimes again this is not so. As a routine the warm specimens from the three types of test diet are best, but even here one has to occasionally resort to feces extract addition in individual instances.

Another method commonly made use of is to study the running bacteriological changes occurring over a week's time in the same tube. For this line of study the fluid media are used, one or two of each being necessary for aërobic study (those studied each day for five), about seven being necessary for the anaërobic (these later being examined on 1st, 2d, 3d, 5th, 7th, 12th and 20th days of incubation).

The object of these observations (and they should be routine with the other procedures) is to observe which bacteria grow and with what abundance, since the rule is that a quick abundant growth of any one or two organisms often has an etiological significance. This is not constantly true, since not uncommonly in true infections an innocent organism would quite outgrow and mask the others, and, therefore, as in all bacteriological observations when in doubt, subculturing is necessary for identification. Following these mixed inoculated cultures along from day to day it is not uncommon to see the bacteriological content change and simplify down to one or two organisms.

By studying from day to day the play of lyses, symbioses and resisting forces present in the different bacteria, biological suggestions are gained that are often well worth while. The symbioses may be manifest as early as between the 24th to the 48th hour after inoculation, although here it must be remembered that the abundant presence of several forms does not positively mean their symbioses. For this deduction the many symbioses of the different intestinal organisms must be understood, because with a loop inoculation of feces into a fresh medium there is nutritive material enough for many forms to grow, even the antagonistic forms.

Again a toxic symbiosis may be present in the bowel, one or more of the grouping not growing at all aërobically or anaërobically as the case may be. Therefore, most often the studies must be carried all the way through with both methods of growing and lengths of time for more worth-while deduction. The lytic changes are best seen after the forty-eighth hour, the less-resisting organisms disappearing first, those more so later. At the end of the observation usually but one or two forms persist, and often these are of etiological significance in the biological make-up. Through the days of these observations subculturing is essential for identification.

Importance of the Multiple Media Inoculation.—The reader must definitely understand that no procedure is more important than the multiple media inoculation presented, and the author hopes that too much reliance for diagnosis and therapy will not be placed on the simple types of procedures enumerated in the early part of this chapter. There is no short-cut or simple method in this work if it is to be done properly. A large number of different media are necessary to be on hand for set-ups in diagnosis. These are selected, added to, changed, or persisted with according to suggestions acquired on the way, but as a rule all are used.

The number is quadrupled for anaërobic studies, and the latter are multiplied according to the number of tubes or plates required to be opened and examined from time to time, there usually being from four to seven. These are added to by more or less subculturing required on the way, and, when in addi-

tion to the standard test diet, the protein and carbohydrate test diets are employed, the whole procedure of several hundred tubes and plates is doubled or tripled.

Truthfully it may be stated that the most common of disorders of mankind requires the greatest length of time, experience, expense and trouble to diagnose and treat properly—there is no short-cut.

Complement Fixation Work.—Of course, as the laboratory studies are continued, straight cultures are being acquired, these being necessary for two main points, namely, biologic therapy and occasionally for complement fixation work. Suggestions on biologic therapy occur elsewhere in this volume.

Regarding complement fixation work for diagnosis some important points may here be repeated. If the biologic studies with stools are well done there seldom is any, and usually no, need for biologic blood serum diagnosis. If any be done, it should be done against the patient's own bacteria isolated by the above methods, and not by stock strains out of the ice-box, the latter of which, in my experience, is hopelessly in error at all times. When done in this way definite results should not be expected in any but the exogenous organisms, and even when these are positive, there should have been much diagnostic biological data on hand on the case so that positive results which merely may represent symbioses are interpretable somewhat near their clinical values.

A large source of error, in all of complement fixation work even when properly done, is that many of the intestinal organisms are non-toxin producers by themselves, and are only toxic in their associations when factors of toxicity in an indirect way. Largely these endogenous organisms are the most important to know about in the case, and commonly they belong to the anaërobic bacteria against which complement fixation work is always practically worthless.

Considering only the aërobic bacteria, or the facultative types both ways, complement fixation work when done as suggested is of value in but 40 per cent. of the exogenous organisms and in error in the other 60 per cent. Straight bac-

teriologic methods are far to be preferred in diagnosis and as handmaids to therapy.

CONCLUSIONS.

By these methods, watching and studying the cultural work most carefully, identification of organisms is accomplished. As they occur, many of the genus types can be separated, tested out separately in side cultural procedures, and one gains a large and varied experience with many suggestions for therapy from the one stool specimen examined to the finish.

A familiar knowledge of the different organisms is essential, not only bacteriologically but clinically as well. The study of the bacteriology of stools is really a most broad type of special bacteriology, one that takes years and much work to become proficient in and one that requires good fundamental training in bacteriology for the start. Familiarity with the symbioses and the associations is a most important part of it, and just the ability to attach importance to findings of this or that organism here and there in the studies is a matter of no small moment. Since biologically there are no two cases alike when everything is considered, and since commonly as many as a dozen pathogenic, saprophytic or zymogenic organisms will finally be worked out as of significance, the diagnosis alone is a maze unless one has had considerable experience with these laboratory procedures.

It is my custom to chart incidence and significance on side notes on the way through the examinations of the organisms identified and those being run down, and giving to each a position in the infection make-up of initial prominence or association values. If the work is well done, one finishes the biologic studies with an organism set-up that suggests the therapy for the start. I say for the start, because with the most perfectly conducted and carefully evaluated examinations, mistakes may nevertheless happen, these to be caught in the subsequent examinations or check-ups of future stool specimens.

What I realize will make this work especially difficult are the following essentials for its proper conduct: A laboratory properly equipped and running constantly on just a few speci-

mens of stool and in which practically no other kind of laboratory work is being done; a worker with assistants whose knowledge of intestinal bacteriology has been built up by experience, time and patience; a combination of this person with that of a clinician, or a clinician with this type of laboratory man, because once the treatments are begun the patient is a laboratory matter and the laboratory is the therapy, and unless these are in a most close connection, errors, failures and limitation of results will inevitably ensue; and, finally, the financial inability of the majority of patients to make even a fair monetary recompense for what is really a service of several months' time, and could not be compensated by only the payment of a few visits now and then. After the clinical sides of the case, in an initial way, had been finished and biologic treatments are being engaged in, what one gives is a service of an advanced laboratory type. For the work to be properly done means practically steady laboratory procedures for four or six months of time.

Of course, even with the most painstaking endeavors, failures will occur here and there because of inability of arriving at the proper biological diagnosis, non-response to biological therapy or anaphylactic matters, and the more cogent ones that exist in the non-coöperation of the patients themselves. On the whole, however, with all that it costs in study, time and expense, the work is well worth the while, and there are no other methods that will produce the results possible of being accomplished.

For data regarding morphology, staining characteristics, cultural methods of examination, etc., the reader is referred to Chapter X, and to standard works on bacteriology, especially those on the enteric organisms.

CHAPTER VI.

EXAMINATION OF THE URINE.

The casual specimens examined at the time of the first examination of the patient in which the specific gravity, pH value, glucose, albumin, and presence of the color toxic products from the intestine, etc., had already been noted in the history. One now proceeds with the 24-hour specimen left at the laboratory at the time of the cold specimen of feces. To present the examinations required in the simplest way the record sheet on following page is employed.

SIGNIFICANCE OF FINDINGS.

The Sulphates.—It will now be noted that the author's¹ modification of the Stokvis-Jaffe test for indicating the presence of indican, urohematin and urorosein of this first specimen is now supplanted by tests for the fixed and ethereal (conjugate) sulphates. Especially important is the relative amounts of the two. The ethereal sulphates are conjoined with aromatic substances belonging to the oxy-group, most important of which are the salts of phenol, indoxyl and skatoxyl. As a rule their amounts vary with degrees of intestinal putrefaction (although not always) and hence this serves not only as a diagnostic link but helps to figure the index of intensity. The amount of total sulphates excreted in twenty-four hours in a normal person varies between 1 and 3 grams, the ratio of the preformed to the ethereal being roughly as 10 to 1.

There are certain conditions in which a relative increase or an absolute decrease of total sulphates occurs. These should be remembered as increased in practically all febrile states, pneumonia, acute myelitis, leukemia, diabetes mellitus, diabetes insipidus, carcinoma, progressive muscular atrophy, pseudo-

¹ Bassler: Diseases of the Stomach and Upper Alimentary Tract, 6th Ed., p. 310.

AUTHOR'S BLANK FOR URINALYSIS.

<i>Physical.</i>	<i>Amts. Found.</i>	<i>Per cent. of Average.</i>	<i>Average Men.</i>	<i>Average Women.</i>
Collected during (hrs.)				
Quantity (Gm.)			1350	1300
Color			Amber	Amber
Deposit			0	0
Odor			Urinous	Urinous
Specific gravity			1016	1015
Total solids (Gm.)			34.19	27.46
Urinary depuration			4.175	4.200
<i>Chemical.</i>				
Ac. terms H_2PO_4 pH 6.5 (Gm.)			1.00	0.79
Total nitrogen (Gm.)			7.43	5.40
Urea (Gm.)			13.29	9.88
Uric acid (Gm.)			1.5	1.0
Ammonia (Gm.)			0.700	0.65
Creatinin (Gm.)			0.96	0.63
Chlorides (Gm.)			6.89	6.93
Phosphates (Gm.)			1.75	1.17
Fixed sulphates (Gm. SO_3)			1.113	0.976
Ethereal sulphates (Gm. SO_3)			0.132	0.106
Neutral sulphur (Gm. SO_3)			0.186	0.200
Sugar (Gm.)			0	0
Sugar (phenylhyd. test)			0	0
Fermentation			0	0
Diacetic acid			0	0
Acetone			0	0
Oxalic acid (Gm.)			0.05	0.05
Albumin (% vol.)			0	0
Albumin (Gm.)			0	0
Urobilin per 100 c.c.			3 mg.	3 mg.
Bile			0	0
Phenol			0.02	0.02
Alkapton			0	0
Lactic acid			0	0
Amino-acid			0	0
Indolacetic acid			0	0
Diazo-reaction			0	0

MICROSCOPICAL.

<i>Organic.</i>	<i>Chemical.</i>
Casts (No. in field)	Amorphous urates
Pus cells (No. in field)	Uric acid crystals
Blood cells (No. in field)	Calc. oxal. crystals
Spermatozoa (No. in field)	Amorphous phosphates
Epithelia Mucus	Triple phos. crystals
Bacteria Yeast cells	Other crystals

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hypertrophic paralysis and some types of eczema. The conjugate sulphates may be noticed in intestinal toxemia (increased), carcinoma (increased), volvulus (increased), chronic nephritis (increased), obstructive jaundice (increased). V. Noorden regarded the elimination of more than 0.3 gram of conjugate sulphates in twenty-four hours as excessive, the patient being on an ordinary diet.

My opinion is definite on this point. With patients on the regular test diet the normal ratio in the male should be 10 to 1 and in the female 9 to 1. Distinct departures from these ratios are commonly of diagnostic significance. Much work has been done on neutral sulphur (derived from sulphocyanides of saliva and cystein) in the hope that an index of metabolism of protein material might be gained, but this has been to no avail.

pH Value.—The pH value is of considerable value (this is best estimated with the urine passed at the time of physical examination and again when the patient comes to the office when on the test diet) in which 6.5 is taken as the normal standard. As is well known it commonly is more acid (lower pH) in many forms of digestive disturbances, especially in the toxemias of the saccharo-butyric groupings. It also is low in fevers, gout, nephritis, leukemia, scurvy, etc.

The Chlorides.—The elimination of the chlorides furnishes a fair index to the digestive powers in a general way, but is of no value in diagnosis in the intestinal toxemias. The phosphate estimation is occasionally a fair index of metabolic disturbance in the lecithin-containing tissues (mostly nervous) and in this connection may serve for suggestion in these ways. As with the chlorides, no diagnostic value in intestinal toxemia can be gained from them. The same may be said regarding urea.

Ammonia.—Ammonia, however, is an important urinary constituent to estimate, but the values must always be judged relatively. Of course, the presence of diabetes, pregnancy and other conditions in which the output would be increased, must be eliminated, included in which are insufficient amounts of fresh air and outdoor activity (the relative factors). When

there is no sufficient reason for it, its increase must be taken as another link in the diagnostic chain of intestinal toxemia.

Normally the amount voided corresponds to from 4 to 6.5 per cent. of the total amount of nitrogen, *viz.*, to about 0.7 gram in the twenty-four hours. When the amount exceeds this, a status toxemia should be suspected, this usually being of the general putrefactive grouping.

Uric Acid.—Uric acid is also an important substance to estimate, because it represents in this connection a metabolic disturbance most often intestinal in origin. When one can eliminate leukemia, in which it usually is increased from the leukocytes, and diabetes, articular rheumatism and anemia, in which it may be diminished, a distinct increase of uric acid above 1.5 grams may be taken as of clinical significance.

If gout is present an increase above this figure is taken as an index of an intestinal toxemia and not as an index of a metabolic disturbance. Gout is not due particularly to an increase of uric acid in the system, and more to the systemic effects of an intestinal toxemia. It, therefore, is not a clinical entity under the term gout, a term that should be discontinued. There are several types of biological errors in the intestines in which moderate increases of uric acid occur. This, therefore, may be another link in the chain of findings.

Oxalic Acid.—The estimation of creatin and creatinin are of no value in this connection, but the test for oxalic acid should be made. This substance is derived from ingested foods and also from the metabolism of the body tissues. The greatest quantity comes from foods and under pathological conditions in the digestive tract as a result of a fermentative process from carbohydrates. The amount normally formed is not more than 2 or 3 centigrams, and this amount on any of the three forms of test diets is negligible. If jaundice, hepatic cirrhosis and pneumonia can be excluded, a daily output of this acid above 0.5 gram is usually another index of an intestinal toxemia.

Albumins.—Nothing need here be presented on the albumins except to call attention to the diagnoses of "functional," "cyclic" and "orthostatic" albuminuria, which are commonly

not states of normal individuals. The terms "physiological" or "constitutional" albuminuria should be eliminated because they are most often evidences or resulting states of some status of a chronic intestinal toxemia. Peptonuria, while it may occur in advanced phthisis, pneumonia, gastric ulcer and post-pregnancy, also occurs in slight degrees in intestinal toxemia.

Important here are the instances of slight degrees of albumin in the urine due to kidney irritation from toxic substances in the blood originating in the intestine. All "trace of albumin" urines and those in which there are slight or moderate degrees of nephritis (in which other tests are also employed, such as concentration, phthalein, etc.) should be studied out, since the majority of these are instances of intestinal toxemia. (Of course, the albumin here is only an indirect evidence of intestinal toxemia.)

Glucose.—It is believed that minute traces of glucose may occur in urines of normal people, such not being noticed in the usual laboratory tests employed for its detection. It is my belief that, excepting under the most extreme conditions of carbohydrate and mainly sugar intake, no glucose should be present in urines, and no matter however trivial, its presence has significance. I am willing to agree that while trivial amounts of albumin may occur normally, this is not true of sugar.

Therefore, following the negative results from whatever chemical test is employed, the use of a polarimeter (saccharometer) is essential in this connection and differentiation with the phenylhydrazin and fermentation test is often required. As a diagnostic rule in this connection I do not employ the 100-gram glucose ingestion test except in diabetes mellitus. Should any sugar occur in the urine from the regular and even the carbohydrate test diet and a true diabetes be excluded, it should excite interest. There are, of course, instances of sugar output in various diseases of the liver (about 11 per cent. in all), alcoholism, phosphorus and other poisoning, some nervous and mental disorders, pregnancy, and heart and kidney conditions, but the main point of interest here is in the so-

called "digestive glycosuria" or galactosuria, which, in my belief and experience, is never a normal status of affairs.

At the time these minor sugar states were described nothing was known of blood chemistry and but little of digestive disturbances. In a large group of the intestinal toxemias increased amounts of sugar in the blood is a common finding, and practically the whole importance of such hyperglycemic states (not true diabetes) is that they are of toxic intestinal origin. I am not able to state any significant connection with lactose, levulose, maltose, dextrin, laiose or the pentoses and glycuronic acid, my experience being limited to the significance of glucose entirely.

Urobilin.—While nothing here need be said of the blood and biliary pigments, biliary acids, cholesterin and pigments referable to drugs, there are pigments of the urine of much significance in this work. The most important of these is urobilin. My opinion is identical with that of Garrod, Müller, Schmidt, Esser and others that this is practically always of intestinal origin. I believe, too, that its presence in urines would not occur without abnormal bacterial action in the bowel.

A great deal of time and effort is being spent on the significance of urobilin in connection with a test of hepatic efficiency and its association with hepatic disease. That it answers as perhaps the best test we have for "liver efficiency" and diagnostically in part with hepatic disease, this whole work will always be incomplete unless the first premises for figuring its occurrence in urines is that in connection with intestinal toxemia. Of its significance in this relation there is no doubt, because in several hundred instances where there has been no evidence of a high urobilin urine on proper laboratory work on stools, there has never been proven the presence of a distinct primary toxemia. Exceptions to this may or even may not be seen in liver cirrhosis, chronic malaria, pernicious anemia, carcinoma, scurvy, Addison's disease, hemophilia and intracranial hemorrhages in which this chromogen may occur. The normal range considered was 3 mg. in 100 c.c. of urine.

Phenol.—Next of interest in this connection is the occurrence of phenol (paracresol), not infrequently found in putre-

faction states, perhaps with no indican in the urine. The same is true of alkapton, which with phenol has informatory importance. The so-called Rosenbach reaction is merely one of a combination of the oxy-products and in that sense is helpful, too.

Diazo-reaction.—Of course the so-called diazo-reaction is of generally known interest in connection with typhoid fever. This reaction, however, is indicative of a mixed infection and may, of course, occur also with measles, scarlet fever, erysipelas, pneumonia, diphtheria, pyemia, miliary tuberculosis, etc. What is not known, however, is that organisms related to the typhoid bacillus may mix with others in the intestine and give positive results. If one can exclude the diseases mentioned, its occurrence (although usually it is rather faint) is another link in the chain.

Diacetic Acid, Etc.—Other than in diabetes mellitus, no significance has come from tests for acetone and oxybutyric acid. But this is not true of diacetic acid, which, when pregnancy, diabetes and drugs can be eliminated, is an index of moment. The same is true of both lactic and indolacetic acids (especially the second mentioned), which should always be tested for and judged significant. Tests are also made for the apparent excess of acids over bases (amino-acids). Many times positive results are obtained in amounts over 0.3, which may be taken as the normal standard of adults.

Ptomains.—Much work has been done on testing for the ptomains cadaverin, putrescin and neuridin, but the amounts obtained were always so small, the tests so difficult, and the results always so questionable, that the tests have been given up as a routine procedure. It is hoped, however, that the chemistry of this subject would develop sufficiency for utilization of tests of these substances in routine ways. Quite obviously when positive findings of the diamins are obtained, they would be a sign of interest.

Leukocytes.—In intestinal toxemia sometimes the leukocytes are increased in numbers. In the cases, however, increased cylindroids and simple types of casts are not uncommon, these probably being an expression of kidney irritation.

The evidence here would be very indirect and not of much clinical value. *Escherichia coli* urines without pathology or infection in the urinary tract also are not judged important, although in these infections of the intestines such urines may have significance.

Specific Gravity.—It is not uncommon to observe urines in the high normal of specific gravity, especially in those whose water intake is habitually low. It has no special significance; the color may be distinctly deeper than in normal, perhaps rather red looking. This has no special significance in itself.

Other Findings.—In closing the subject of urinalyses in connection with the diagnostic appreciation of intestinal toxemia, there are many indirect signs of value. None of them is useful in definitely specific biological ways and the evidence gained is to be taken as additional evidence of the correctness of the diagnoses in a blanket sense and not in a specific detailed way either completely diagnostic or therapeutic. The urinalyses, however, require the utmost detail in the highest types of quantitative chemical procedures, the ordinary ones commonly performed in office and most laboratories being of little or no value and largely leading to errors, usually in a negative sense, but sometimes positive.

The findings should be recorded individually and it is best to draw no conclusions from the urinalysis and the cold feces work, until the technical work on the feces especially has been finished or carried far enough for definite results. The reports may then be assembled and the findings built up and correlated for conclusion.

Bacteria.—It often is of importance to culturize urines for the identification of the important and especially the infecting types of bacteria. Before significance is attached to the so-called *B. coli*, this *Escherichia* should be worked out by culture methods, this being necessary because of the many innocent forms of these organisms in urines. In this, of course, clean specimens of urines are required. There are a number of infecting types of bacteria found in urines, the organisms arriving there from the intestinal canal in which their presence can also be proven.

CHAPTER VII.

EXAMINATION OF THE BLOOD.

It is unfortunate that the examination of the blood has not advanced to the estimation of constituents regarding which practically nothing is known as yet. It is logical to believe that in infective conditions the blood must contain constituents, tests for which would be most valuable in diagnoses. Among these would be methods of determining quantitatively indol, indoxyl and phenols in the blood, all important in this connection. In the absence of these, and confining one's work and observations to those that are known, not much of real value in this connection is obtained from estimating the well-known blood constituents.

BLOOD CHEMISTRY.

Sugar Estimation.—Of course findings pertaining to the diagnosis of renal diabetes (normal amounts of blood sugar) and diabetes mellitus (hyperglycemia), gout (uric acid perhaps increased), nephritis (urea and creatinin increased), prostatic obstruction (increase of urea in estimating operation risk), pernicious anemia (hypercholesterinemia) may have a relative significance in connection with the diagnosis of intestinal toxemia, this depending upon the case. There undoubtedly is in sugar estimations a factor of considerable importance in connection with a large part of the group of the fermentative intestinal conditions.

In what may be loosely classed as those of the saccharo-butyric group, moderate degrees of hyperglycemia are almost uniformly found. These occasionally show a trifle of sugar in the urine (usually not so), the patients being non-diabetic in any strict sense; and the estimations of blood sugar found in this connection have often served as a sign of interest in the blood sugar being higher than normal. In certain endocrine conditions even when secondary or contributory to intestinal

toxemia, higher than normal blood sugars may be found (when hyperthyroidism is present especially). As is known, the sugar content may be low in myxedema, cretinism, Addison's disease, hypo-adrenalism, pituitary disease and muscular dystrophy in more or less fundamental ways. The sugar content factor then may or may not be of value in a suggestive sense.

Carbon Dioxide Combining Power.—The carbon dioxide combining power may or may not be of value. Apart from the ketosis of diabetes, the normal balance of slightly alkaline reaction which is maintained through the influence of the bicarbonate, phosphate and proteins of the blood may be interfered with so that figures from 75 to 50 CO_2 capacity in 100 cubic centimeters of plasma may be changed and yet there is no true diabetes or nephritic acidosis present. It is probable that this occurs, not because of the presence of acetone bodies, but by interference with the bicarbonate and deficient excretion of acid phosphate by the kidneys.

More especially is this observed in the so-called "acidosis of children," in which the figure may go as low as 22. Parenthetically it may here be recorded that the "acidosis of children" is generally caused by intestinal toxemia and it is my suggestion that that term be not continued and that the whole subject be placed upon a proper scientific basis.

Hypercholesterinemia.—Moderate degrees of hypercholesterinemia are frequent findings (above 0.14 to 0.17 per cent. for the whole blood) and possess a value when that due directly to arteriosclerosis, obstructive jaundice, early stages of malignant tumor or pregnancy can be excluded. Cholelithiasis and certain skin conditions in which the cholesterol is higher than normal are judged more in the light of intestinal toxemia than that the increased cholesterol is due to these conditions (not resulting states therefrom). In the instance of anemia (in which the cholesterol is generally low) the degree of anemia must be taken into account relatively with the cholesterol content. In this way low figures may nevertheless have significance.

Uric Acid.—No worth-while experiences have been had with the blood chlorides, my opinion being that they are not

of value in this connection. The same is true of creatinin. In uric acid, however, we again have a factor of importance. There is no blood constituent so readily raised by intestinal toxemia as uric acid. It is known that one may meet with high values in nephritis, leukemia, gout and arthritis, and more or less changes can ensue from the high intake of certain types of food (proteins) and drugs. In amounts above 2.3 mg. to 100 cubic centimeters of blood it is one of the most significant of the blood signs.

It is essential to state here that to do a blood chemistry properly is one of the most technical of procedures. One has but to submit a half of the same specimen to two different laboratories to prove this. There are many men and laboratories doing blood chemistries and but few of them accurately in my experience. Since the definite findings in blood sugar, carbon dioxide combining power, cholesterin and uric acid are increased in only moderate amounts in these intestinal conditions, great accuracy is required or the work is useless.

The Icterus Index.—The determination of icterus index is employed clinically in connection with the detection of jaundice states. If the figure of 4 to 6 is accepted as the normal standard, this test is of much value in the work. In this connection the bilirubin content of the blood may be considerably increased with perceptible jaundice never occurring. This type of so-called "latent jaundice" has been studied in connection with gastric and duodenal ulcers, gall-bladder disease, malaria, jaundice states, pneumonia and diabetes mellitus, in all of which it usually is moderately raised.

But moderate degrees of bilirubinemia are a distinct aid in the diagnosis of intestinal toxemia, and represent the commonest cause for its presence. Like urobilin being believed to be a worth-while test for liver efficiency (when it is a poor one for that but a good one for intestinal toxemia), so the icterus index is a better test for an intestinal state than any of the other clinical conditions in which it is deemed valuable, although its value in jaundice, especially in a differential sense between the hemolytic and non-hemolytic anemias, is most worth while. In my work a value over 6 is taken as significant.

Hemoglobin.—The estimation of hemoglobin is of some value, because practically all of the simple anemias are due to intestinal toxemia, the anemia occurring by lytic bodies affecting the erythrocytes directly (largely the sulphur-containing substances), these seeping into the blood from the small intestine mostly and further depressing the functions of the red blood cell making organs. Since there are other factors in the production of simple anemia, a lowered hemoglobin should be judged in an analytical sense. This, however, is not true, in so far as pernicious anemia or the "border line" anemias are concerned, they all, in my opinion, being largely bound up in states from an intestinal toxemia.

Complement Fixation.—Of interest now in the diagnoses are the phenomena of complement fixation. Since Bordet's discovery of the hemolysins, much work has been done to utilize this method in bacterial diagnosis. While its value is accepted in connection with the Wassermann reaction, the work in connection with bacteria estimated in antigenic stimulation in so far as the intestine is concerned has been practically of no dependable diagnostic value. In the many organisms of the intestinal canal with the many degrees of activities from very active all the way down to complete absence of activities, any test of the blood to diagnose this or that infection is more or less doomed to failure from the start. As has been stated, many of the most important of the organisms develop very little complement fixation antibodies; others, especially those of the typhoid-colon-dysentery-cholera group, being distinctly more active. It has been thought erroneously that under proper technical conditions the complement fixation reaction is a more accurate means for studying biologic relationship than the agglutination and precipitin reactions. My belief is that none of them are of signal diagnostic importance in connection with intestinal toxemia. The tests, however, are of value in syphilis, gonococcus, glanders, contagious abortion, typhoid and some other diseases.

Agglutinin Reactions.—Practically the same, although not to as definite extent, may be stated regarding the agglutinin reactions. In the first place, the majority of intestinal

organisms are endogenous and do not produce exogenous toxins. In those of the typhoid-colon-dysentery group some diagnostic suggestion may be obtained, although because most intestinal toxemias are mixed infections (commonly of exogenous and endogenous organisms in symbiotic association) the identification of one would give no significance of the other. Still one might repair to this method at times for further elucidation in clearing up a biologic puzzle and occasionally suggestion is accomplished. If very carefully controlled and estimated in a relative sense, occasionally suggestive data are obtained. Normally serums of adults agglutinate typhoid, paratyphoid, colon, pyocyaneus and the dysentery bacilli. For the purpose of this work one must have on hand a large supply of the intestinal organisms, or various immune serums if the patient's microorganism is employed. The organisms used in my work comprise practically all of the isolated organisms and strains that can be kept viable.

It has been my experience that, briefly expressed, this method of testing is of some diagnostic value in about 15 per cent. of all instances, and of very slight value in about 30 more. But, when positive, the result must be judged on other findings in addition; when relative, no deduction should be made, and when negative, positively no deduction made one way or the other. The test would be of no value excepting in exogenous infections and then always judged doubtfully. In the endogenous organisms it is useless. In judging parts of associations it has a value now and then.

Bacteria.—The usual methods for proving "positive bloods" for bacteria are well known. Since these are not efficient to prove the presence of many of the less pyogenic bacteria, anaërobic, longer period, and special procedures are required to prove their presence in the blood. In these the cultural methods already suggested would prevail, it being necessary to constantly keep in mind that many of these organisms are of low virulence and difficult to grow. They are found to a far larger extent than is known or believed, their presence in the blood is generally from the intestine, and their importance in clinical states manifest.

CHAPTER VIII.

INTERPRETATION OF SYMPTOMS.

GROWTH OF MEDICAL SCIENCE.

In the early days of medicine all was empirical. As the centuries passed, anatomy, physiology and other fundamental departments of medicine developed for the interpretation of symptoms on pathological bases. Since many disorders of the one organ had the same symptoms, three movements then evolved:

First, the advent of study of efficiencies or deficiencies of the various organs, such as the heart, stomach, kidneys, liver, etc., for which many tests have been devised and of which only a few are clinically useful.

Secondly, the attempt of marshaling symptom complexes in character leads toward definite diagnosis, and, thirdly, the advent of apparatus for examinations, such as the sphygmomanometer, the x-ray, etc. All of these have been of service, yet there always existed an unsurmountable gap. This today is being filled in partly by the development of the knowledge of man, his reactions to disorders and diseases, and by biology and the subjects allied to it.

There is no more interesting subject in medicine than the fundamental one of etiology. To the real student, the causes of disease occupy the status of our greatest interest today, and the transformation from the therapeutic type of medical man who held therapeutics to be the fundamental of medicine, to the man of recent times whose emphasis is on diagnosis is happily placing medicine on a more substantial basis. This etiological work has been actively engaged in by numerous men, most of whom and their work are hardly known by the therapeutic type of physician, and thus much of the best work in medicine has received but scant attention. Nevertheless, little by little here and there, in contributions appearing most often in publications read almost entirely by the studious type

of physician, these workers have plodded on. From the efforts and labors of these biologists, occasionally some ray of light shines through and penetrates into the work of the therapeutic types of today. Most often, however, it is met with lack of understanding, by criticism, and far too often is totally neglected. None among us are more sensitive to criticism than a pioneer, who, all on fire with enthusiasm over some new piece of work, presents it and meets with lack of understanding, criticism, or its neglect. And when this man is a laboratory worker or some other type of biologist, and the practicing side of the profession makes their onslaughts, more than one of our more silent workers become crest-fallen and broken in spirit.

The future of medicine is largely in the hands of our strictly scientific men, because the gaps to be filled in (and they are many) are essentially all in the hands of these ill paid and silent working brothers, who, in my opinion, should be encouraged and respected in every way. This is important because there is no doubt that the developments of the future will be largely in the biological side of our work, and, however we may feel opposed to this, the only real advances in medicine of the past few years prove this to be the fact beyond any question.

HEREDITARY FACTORS IN LIFE.

On some of these matters it may briefly be offered that the studies of human biology have proven that congenital malformations when unfavorable show fundamentally that man is subject to the laws of natural selection in one form or another, and that many of these variations are limited by death of the individuals bearing them.

The immigrant stock in reproduction and evolutionary survival show a superiority to native stocks. This is proven in the original and in one or two generations of them, compared to our truly native New England stock, which is passing away. The immigrant stock stands city life and resists infections better than the native form, and therefore are less prone to infections and surmount them better. The native stock on

the other hand seems to do better under rural conditions than the immigrant. This is a matter of vitality and immunity resistances that has some value in understanding medicine and mainly intestinal toxemia today.

Biologic studies suggest that the physically hard-working person, whether indoors or outside, before the age of forty is quite as well off in a general way as those employing less effort, but after forty or forty-five, man shortens his days in proportion as he performs heavy physical labor. Added to this lowering of the vital index through physical work, are the factors of exposure, foods, rest, etc., all of which when neglected make men over forty-five more prone to the inoculation and the effects of infection.

Duration of Life.—There is no doubt that in man there is an enormous variation in the duration of life both intra- and inter- racially. It is believed by biologists that man is the longest lived of all mammals. Whether this be true or not will not be proven for some years, but in the scale of longevity of animals Pearl¹ has rated man in length of life to between one and one-half to over one hundred years. In the study of this problem there is no generally valid, orderly relationship between the average duration of life of the individual composing a species and any other broad fact now known in their life's history, their structure, or their physiology. We as physicians agree that natural death as distinguished from accidental death is preceded by definite structural and functional changes in the body. This is seen in connection with disease as well as in senescence. The study of the latter is not going to be of much value to us as medical men in the understanding of disease. Certain significant cellular changes take place not only as a part of advancing years but definitely before that time for biologic reasons. There is no doubt but that a break or discontinuity in the progression of life comprises a subject of much interest to a medical man.

Some years ago Metchnikoff held that death was the result of intoxication arising from the absorption of putrefactive

¹ Pearl: *The Biology of Death*, p. 22.

products of the activity of intestinal bacteria. This is only partially true, since life under completely aseptic conditions may be terminated in the usual time. It, moreover, is also not true as connected only with the putrefactive products of activity of intestinal bacteria. There are many toxins capable of being resorbed in the general circulation which are in no sense bound up with putrefactive activity in the intestinal canal, and it is particularly from these toxic substances that the main causes of disease ensue.

Another fact is that the determination of the degree of longevity, and even death itself, is inherent in the innate hereditarily determined biologic constitution of the individual and the species, but this is constantly being assailed and largely can be influenced for weal or woe. It is my opinion that the subject of longevity and disease in man is based not on any one fact but in the interplay of a number of factors incidental to man in the course of his active existence. These comprise factors of nutrition in early life; the bacterial infections of the young; the crossed sex characteristics with their instabilities, and the various abnormalities in the sequential development of the endocrine organs; all of these operating before the adult years are acquired. From the adult years onward, biologic changes occur according to the age of the individual, the period of adolescence, middle age, or senility, and with these are factors pertaining to the sex of the individual. With these there are other environments such as occupation; temperamental types (depressed, etc.); the climate under which the individual lives; the amount of fresh air he breathes, the sunshine, sanitation, etc.; his routine of life; the chemistry of the water he drinks, the food he eats; his amount of sleep, exercise, etc., to which are added the various bacterial diseases he acquires, certain food allergies, and to a great extent the biologic status of affairs of a running type in his intestinal canal.

Beginning with the hereditary factors, the environmental factors also have to be figured in the individual who is before you as to his morphologic make-up, his immunities to infection, his psychology, and his physiology. This may be expressed in the following schema, which is a modification of Draper's, in

which the lower part represents the patient before you. While this is academic, it often is helpful in understanding the complaining individual who presents himself with any disease or disorder, the etiology of which is obscure or unknown, and perhaps the symptoms of which are difficult of summary in terms of a clinical significance without such estimations being figured in the examinations and conclusions.

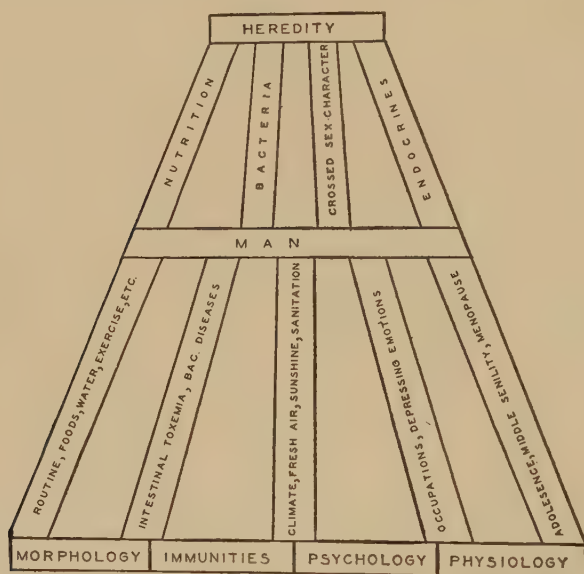


Fig. 5.—Environmental Factors. (Modified after *Draßer*.)

THE ENVIRONMENTAL FACTORS.

It is not possible in a work of this sort to debate various matters pertaining to the schema offered. Their enumeration is all that is necessary to convince one that each of them has a signal importance in medicine, and one needs but to study the histories of various patients, as to the starting time of symptoms and how they accompanied or followed various conditions, to be convinced that man is constantly subjected to environmental onslaught; and his reactions to and relative freedom from disease are conditioned upon a number of factors.

The establishment and the event of disease is commonly conditioned on the exciting or negative factors which affect

him throughout the years. After adult years, however, the major factor, or factors, is bound up in infections and, of these infections, the most common in man are those of the intestinal canal which often run on for years without the production of any symptoms, and when they do occur, these symptoms are commonly remote from the intestines themselves, perhaps involving some distinct essential organ. It has been short-sighted of us in the past to direct our mind only to the organ affected rather than to the etiological cause or contributing factor that has played significantly in the production of the disease.

Functions of the Body Cells.—It must be remembered that the human body is built up of millions of cells. These cells in different organs have more or less specific functions, but all of them are dependent upon the general blood stream for nutrition. Since blood contains many more constituents than is suggested by the present-day methods of blood chemistry, and is quite as much of an absorption tissue as it is one of nutrition or one of elimination therefrom, the various cellular structures of the body by virtue of their chemical make-up and function are amenable to substances contained in the blood acting unfavorably upon the cells. Highly specialized tissues, such as that of the nervous system, heart, liver, kidneys, blood-vessels, etc., show from different toxic bodies absorbed from the intestines, that they may and do have selective properties to toxins. Only in this way can many of the events of disease be explained, and only in this way can much pertaining to functional disorders be understood. This part of medicine may continue to be neglected because some substance in the blood here and there cannot be identified, but with the disease or the condition on one hand, and the study of the biology of the intestinal canal on the other—especially when the latter can be corrected or benefited, thus bringing about desirable effects—much that is obscure in medicine today can be understood and explained. It is because of my experience with these intestinal conditions which, when benefited, have brought about such remarkable results that I believe that the

etiological connection is commonly complete and proven, whatever the argument against it may be.

According to the work of Koessler and Hanke¹ the products from intestinal bacterial activity have been distinctly advanced. It is more than possible that certain of the amino-acids, alone and from the combined products, have selective actions on certain highly specialized tissues of the body. This work has a rather extensive literature and is in large part definite enough to be accepted as true. Among these may be mentioned the substances histidine, histamine, imidazole propionic acid, imidazole acetic acid, methyl imidazole, lactic acid, acrylic acid, alanine, arginine, glycine, glutamic acid, tryptophane, cystine, tyrosine, tyramine, polyphenols, aromatic hydroxy-acids, and no doubt many other toxic amines as well as, in addition, toxic products from carbohydrates and fats. This no doubt explains one of the ways of disease and disorder production. Whether these substances affect the tissues directly in ways toxic and destructive to function and anatomy, or that the tissue reactions to them are the more important is not known, and it will be years before this is properly understood. But with the proof of the intestinal infections on the one hand, and the multiplication of the same results of dysfunction and disease in identical organs on the other, the proof is quite conclusive to me.

Disease and Health in Childhood.—A few general matters pertaining to the biology of man have been presented. We all agree that the human being is born with certain characteristics transmitted from parents which may or may not be operating during the life of the offspring. Taking the infant child as possessing certain hereditary factors, we can begin a consideration of this subject with matters of nutrition. It must be evident that with the exception of the exanthematous diseases of childhood, the pediatrician's work lies almost entirely in matters pertaining to digestive disorders, both in the control of digestive conditions and in the general subject of foods to maintain the processes of life and to meet the require-

¹ Koessler and Hanke: *Jour. Biol. Chem.*, Oct., 1919; Sept., 1920; Jan., 1922; Apr., 1924.

ments of development. A very large percentage of cases of intestinal toxemia in the adult begin in these early years and are due to infections acquired in infancy and childhood.

In addition to the subject of diet and food, pediatricians must go into the biology of this subject, inasmuch as they have recently developed an interest in not a few of the other gastro-intestinal conditions seen in the adult, these being present also in the child. Of late years, largely through the efforts of Dr. Charles Kerley of New York, the subject of the gastro-enterology of the adult has been brought down to the growing child, and now ptoses, dilatations, chronic constipation, ulcerative conditions, and even gall-stones are becoming interesting to the pediatricians. Since in a considerable number of instances of intestinal toxemia in the adult we can clinically trace symptoms back to the tender years of life, another responsibility is placed upon pediatricians to do biologic work in connection with their young subjects, in that way doing prophylactic work of clearing these infections or modifying them before the adult years are reached.

In this connection the incidence of the infectious diseases of early life undoubtedly has an effect on the conditions of the tissue of the grown adult. These are represented in the exanthematous diseases, but more particularly so in the fact that, after the first year or so of life, the human being must acquire the immunities necessary for his or her protection in their latter-day existences. After the birth of the child, and usually for a year or two, the transmitted immunities from the mother protect the child from infection. Since after the second year these are used up, it is necessary for the child to build up its own immunities. This is the time of the usual presence of digestive disturbances, and it is in connection with this amenability and the lack of immunity bodies in the circulation that the child is especially prone to infections of the intestinal canal, which infections can and commonly do persist through the lifetime of the individual.

Sex Characteristics.—The study of human beings proves that but few of us are Simon pure in sex characteristics. In

clinical work the female with distinct masculine characteristics, and the male with feminine characteristics, are commonly seen. It is not possible to go into this subject very deeply, but it should here be recorded, that where definite crossed sex characteristics exist there is usually an instability or an under-endowment in the nervous system characterized in a running way in the individual. This often allows a hypersensitiveness and intense reaction to various environmental causes, and commonly the complaining is greatly intensified beyond the cause or the condition. In gastro-intestinal work the neurological make-up of the individual is an important factor, and any fundamental condition which is permanent, like crossed sex characteristics, requires attention in the understanding of symptoms.

The Glandular System.—Without going into the subject of the physiological and pathological correlations in the reciprocal action of the ductless glands or discussing the many questions of dysfunction, we have to agree that these glands have a distinct influence upon growth, configuration of the body, regulation of metabolism, effects upon the nervous system, etc. We also have to admit the importance of a high-grade hypoplasia of the sexual glands at an early age leading to tall growth, slender build of the skeleton, and certain characteristic proportions as well as delay in the closure of the epiphyseal junctures, all of which are mostly operative at the time of puberty.

Absence or high-grade diminution of the function of the thyroid gland leads to dwarf growth and other morphological characteristics. The absence of the hypophysis in early years also leads to inhibition of growth and other morphologic factors. Certain conditions also develop in hypofunction of the sexual glands, and the whole glandular system has a considerable influence in the regulation of metabolism of the growing child. The latter is seen in the carbohydrate metabolism, that of the protein, and in certain other matters such as the respiratory gaseous exchange, heat regulation, and certain factors influencing the water economy. From birth to adult man, the subject of endocrinology occupies a most important

part in morphological development and in the various types of dysfunction. The mono- and the peri-glandular conditions should be studied in connection with this subject, as well as the endocrinologic side of hyperthyrosis and hypothyrosis, especially the partial type; with aparathyrosis or hypoparathyrosis; the diseases of the hypophysis and dysfunctions of the pituitary gland; Addison's disease; the excess symptoms of suprarenal conditions, especially those of tumor formation; status lymphaticus and status hypoplasticus; eunuchoidism; as well as several others distinctly belonging to the domain of diseases and conditions of the ductless glands.

The author wishes to state here, however, that while in all probability the intestinal toxemias of the young do not have the damaging effect in the production of disturbances of the ductless gland system that they have in the adult, they do become operative in distinctive ways as the child is emerging into adolescence. These effects upon the glands are mostly produced by toxic substances circulating in the blood and having a distinct selective effect upon gland development and gland function. When adult years have been reached these deficiencies of function have become more or less definite and are often discovered in clinical work, and while suitable endocrine therapy does benefit some, in the vast majority of instances the glandular dysfunction is due to the intestinal toxemia and remains so, no matter what the glandular therapy may be. In my cases there have been so many instances of deficiency of glandular function wherein a resumption of the gland function to normal occurred after the removal of the intestinal toxemia, that it is interesting at least that endocrinologists do not realize the connection that exists between the biology of the intestine and the study of the internal secretions.

In my own work with the adult the use of glandular therapy has become less and less frequent, and insofar as their use (in the belief of today) in benefiting neurological conditions is concerned, almost the entire subject is not one of glandular therapy but the treatment of the underlying intestinal toxemia, the toxins of which have a depressing effect

upon the glands. This toxemia when removed brings far more benefit to the functional neurological condition than any amount of glandular therapy could accomplish. And more than that, the benefits are substantial while those of glandular therapy when accomplished are mostly transitory.

Benefits of Regular Living.—A normal routine of life is essential to most people, especially in middle age, to preserve a status of health. Those who keep irregular hours, eat meals at irregular times, and hastily, with the intervals between them not reasonably spaced, who retire at irregular times so that there develop periods of definite fatigue, all these are lowering their physical and vital forces, and all have their effects.

Improper food—by that I mean that of insufficient nutritive value as well as excess eating, partaking of too many vegetables and fruits, or the eating of too much protein food—have a connection, not only in the production of general body conditions, but a more or less inimical effect on the biology of the intestinal canal. The drinking of too little water or fluids to meet a full elimination from the kidneys, neglect of the skin and rebreathing of expired air has to do with general body states, and very commonly also deficient elimination of substances from the body which arise in the intestinal canal. The taking of too little exercise, such as in those who pursue a sedentary existence, invariably means a stagnant metabolism, especially in the tissues, deficient urea formation, excess uric acid, and the lowering of the general oxydase ferment products throughout the entire body. It is also a factor in producing deficient conjugation of the toxic substances into chemical products which would be less inimical and easier of elimination than the toxic substances themselves.

Immunities.—The subject of the intestinal toxemias then comprises also a consideration of the major parts of all of the environmental factors of man insofar as they affect running immunities, and since this volume is devoted to the intestinal phase of this subject, they need not be discussed further here. Important to mention especially, however, are the bacterial diseases which occur as acute and chronic infections in various parts of the body, such as influenza, pneumonia, typhoid fever

and the many others that are found described in the book of medicine.

Recovery from many of these means the accomplishment of immunity against the infection, but not infrequently this immunity against a definite disease or a definite infection is accomplished at the expense of other running immunities that may be present in the blood at the time. While then a specific immunity would be gained against the disease from which the individual has recovered, the diminution of other immunities renders him liable to mixed infections occurring at the time of the original infection or to others occurring subsequent to the original one. Many, many times this susceptibility to other types of infection has occurred, and of such disorders there is none more cogent in importance than the blood immunities holding intestinal toxemia states in control and then being modified or lost.

Climates.—All physicians are acquainted with the beneficial and damaging effects of various climates. On this subject there is much that could be said, but the reader needs but to think of his own personal experiences, and those by which he has been able to accomplish benefit for his patients, to realize that climate is an important environmental factor of man. Also of vital importance are fresh air and sunshine. There is nothing more sure about the entire work of infections, whenever they may be found, than that individuals who live much in the fresh air are less prone to infection than those who lead indoor existences, and that in recoveries from infectious disease and convalescence, fresh air and sunshine are important factors. Especially when toxic bodies are circulating in the blood stream, by the breathing of an abundance of fresh air, they are bound up in chemical union, an important constituent of which is derived from the oxygen of the inspired air. As has been stated these conjugated substances are less inimical to the tissues than the toxic substances themselves, and further, they are far more easily eliminated from the body.

Sunshine, perhaps the most effective of all antiseptic agents, is as important to man as it is to vegetable life. It increases elimination from the skin, and there are many toxic substances

in the blood which only the skin can eliminate, or at least eliminate in the largest quantity. Also important is the antibody stimulating effect of sunshine on the skin.

Sanitation.—Regarding sanitation there is much that could be said. It must be evident that with good sanitation the foods not only are more wholesome but they are more free from bacteria. Man very largely infects his intestinal canal by way of his mouth with organisms on foods that he has ingested. These, of course, are more commonly found in foods that are taken in their raw state, such as various vegetables, salads, fruits, etc. Likewise sanitation is important in connection with water and the control of contractable infections that may be carried into the mouth or breathed into the lungs.

Occupation.—Of late years, in medicine, occupations in connection with various diseases and conditions have received considerable study. These need not be gone into here, excepting to state that those bound up with contacts with various chemical substances, either in elemental or combined forms, diminish the resistance of the body to infections of all types. These are too numerous to dilate upon, the broad statement of significance being sufficient, that occupations comprise an important environmental factor of man. Also of importance in this connection are the various depressing emotions, such as anxiety, grief, fear, suspense, etc. They all have effects upon the running status of nervous system, and not infrequently depress the functions of body tissues so that the general health is impaired and perhaps the resistance lowered.

Age Periods.—It is a well-known fact that in the various ages of man, the status of susceptibility to different diseases more or less operates. As a rule, the younger the individual, the greater the susceptibility to infectious diseases, while those who are middle-aged usually have acquired more or less immunities, and those who are older certain resistances of tissue that would be helpful. In this connection, of course, should be considered the menopausal states of women in which various metabolic phenomena and nervous conditions ensue. These need not be entered into extensively here and are merely mentioned for recollection.

With the knowledge of the etiology, pathology, symptomatology, prognosis and treatment of the various disorders a physician has to contend with, one must now study and evaluate the hereditary and environmental factors which operate on the child and during the course of adult years, to have an understanding of the individual who has the disease, as well as to understand the disease in the individual. The knowledge of these factors presents the answer for certain morphologic characteristics of the individual, certain immunities or lack of them that would be present, the psychologic make-up of the individual, and matters pertaining to his physiology as to whether it is slow, average or fast. All of these are important for understanding not only any phase of medicine in diseases and disorders, but especially for the understanding of the intestinal toxemias.

The reader may wonder why the author has presented so fully the subject of environmental factors in connection with intestinal toxemia. The main reason of this is that errors in them influence immunities, locally in the cells of an organ and generally throughout all the cells in the other organs of the body. Some of these factors build up immunities of specific kinds at the expense of other immunities. The majority, however, are immunity-robbing factors, and as immunities are not built up or become deficient when they had been present, intestinal toxemias are the more significant in the production of disease and conditions. In good general health, with the immunities sufficient, chronic intestinal toxemia is practically nonexistent, and this is true in constipation, whatever the diet. In this same way, therapeutically, whatever treatment is engaged in which has to do with benefiting physiological action of an organ or the body as a whole, it increases resistance to all infections and grants what may be termed a physiological immunity. This is the reason why there are so many different types of treatments that benefit clinical conditions, sometimes for considerable periods. But even here the individual simply "gets on top" of his toxemia for a time, the infection usually continuing and the production of havoc going on in a more moderate way. The environmental factors are handmaids assisting in break-

down and in cell change, and any methods of treatment that assist in the control of these factors have a place in the tissue side of this subject. Obviously, to give all of these is not necessary in this volume. Following the schema presented with questions on the various matters, building up here and correcting there, and with the ordinary therapeutic measures and perhaps some special procedures, answers for the control and benefit of the environmental conditions. One must always remember, however, that these are not the total therapeutics of the subject of intestinal toxemia; control of the infection is absolutely essential, and this can only be accomplished by accurate biological diagnosis and therapy.

VARIOUS WAYS IN WHICH INTESTINAL TOXEMIAS AFFECT THE BODY.

We have been presenting matters pertaining to the biology of the human being, the heredity and the environmental factors of man as they course through life and as they figure in the particular patient, with suggestions as to the sensitivity of different organ tissues to the selective effect of the circulating toxins. What is necessary now is to explain in some way why it is that in two individuals with the same infective type of intestinal toxemia, tissues in different parts of the body should be affected. In one instance perhaps it might be an eye or a heart condition, and in another it may be one of the organs of the digestive canal or kidneys, and in another elsewhere in the body, perhaps even as remote as the circulation in the lower extremities.

The Vegetative Nervous System.—One must agree that the vegetative nervous system is the paramount system in the control of body function. Over the vegetative nervous system the ductless glandular system acts in important control. This system is made up of a number of different parts which are more or less interwoven. It would serve no purpose here to enter into a discussion of minute detail of the various known factors pertaining to the vegetative nervous system and the internal secretion of the chromaffin tissues.

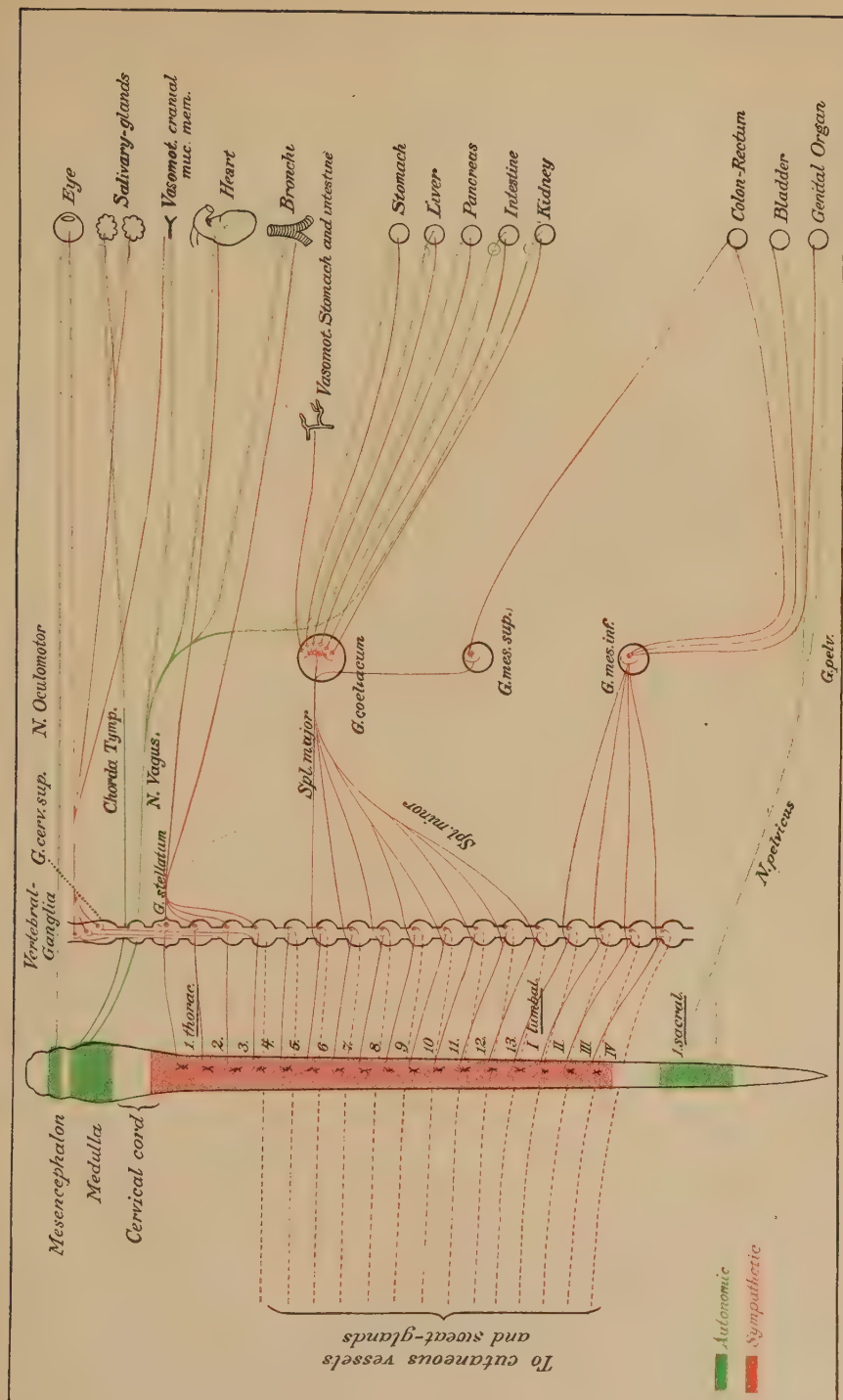


Fig. 6.—Schema of the Vegetative Nervous System. (Reproduced from Falta's "Endocrine Diseases," edited by Myers, after Myers and Gottlieb's "Die Experimentelle Pharmakologie," etc. The autonomic innervation is colored green, the sympathetic red.)

Relationship of the Autonomic and Sympathetic Nervous System.—The above diagram suggests at a glance the inter- and intra-relationships that exist between the autonomic and sympathetic nervous system, and I need but to state that according to the portion of the autonomic or sympathetic nervous system affected, that is, in whatever the level the ganglia are involved, the same toxin perhaps operating on different sections and parts is capable of producing symptoms in widely distributed organs.

Up to the present time it is believed that the ductless glandular system almost entirely presides over the function of the autonomic and sympathetic nervous system. This is only partly true, and as accepted in medicine (even should the biologic and hereditary factors be omitted) falls short in explaining not only the many irregular states and conditions in the nervous system, but also those of the various essential organs. It is my opinion that the ductless glands singly and collectively have a paramount effect upon the functions of the body, but it is also my opinion that the generally accepted belief that all of these conditions start from the glands is only half of the story and the significance.

The energies of the ductless glands are dependent upon blood for their nutrition, and to a great extent according to the presence of toxic factors in it they are affected inimically in their function. These toxic factors are really paramount in the significance of the functions of the ductless glands, and are of first importance in connection with study of all that pertains to the ductless glandular system. It has been proven to me many, many times that when the individual is toxemia-free so far as the intestinal canal is concerned, and there are no hereditary conditions of the glands, the ductless glands are perfect in function, and in individuals who are not toxemia-free one should expect irregularity in the function of these glands. To begin with the significance of the ductless glands on one side and to end with their significance in the production of disease and conditions on the other, is to make the journey of understanding in more or less hopeless circles.

One needs but to follow the simple subject of vagotonia alone to prove that this is the fact, and this is the clinical picture that presents itself frequently in gastro-intestinal and almost endlessly in intestinal toxemia work. To understand the endocrine functions, the significance of the intestinal field in the production of conditions having to do with internal secretion is of importance and puts an entirely different aspect on endocrinology. Much time has been spent in the study of vagotonia in conditions like pylorospasm, hyperacidity, bronchial asthma, spastic constipation, gall-stones, gastric crises, etc., and nobody seems to realize that the vagotonia is a state resulting from an intestinal condition and that idiopathic conditions of the gland are not the beginning or the real cause of the vagotonia in an important sense. It is believed that vagotonia exists more markedly in the young and is an expression of an inferior constitutional make-up, but this latter is largely a statement of belief that means nothing.

I desire to state in conclusion that while there are hereditary factors of significance in the understanding of ductless glandular diseases and disorders, the most important and the most numerous are not hereditary in make-up, but arise from an onslaught upon the sensitive ductless glandular system in the course of our existence; that the development of the short-sighted academic side of this subject has been in denial of the fact that the vast majority of individuals would have a proper ductless glandular system if the environmental factors of life had not operated inimically upon them; and that the most important of all of these environmental factors is that of chronic states of intestinal toxemia. The study of internal secretions will advance better in understanding and handling when this part of the biology of man is taken into consideration—not before.

It is, therefore, according to the portions of the mesencephalon, the medulla and sacral part of the autonomic centers affected, or that of the thoracic or lumbar in the sympathetic, or on the fibers from them to their distributions, as to whether eyes, mucous membranes of the head, heart, lungs, stomach, small intestine, liver, pancreas, colon, rectum, bladder, genital

organs, etc., are the locations of symptoms and conditions due to the toxic factors.

Operating constantly in the production of dysfunction of these organs and tissues, continued or permanent disorder or disease can be brought about by vasomotor disturbance and direct effect on the specialized tissues by bacterial toxins and resulting resisting tissue toxic substances. These facts are most important, and one needs but to identify an inimical intestinal bacteriology which is removable or controllable to prove that this is a most important matter in the control of a large part of the important gaps in medicine.

Other Pathologic Factors.—Added to the above factors and distinctly of a direct contributing nature, are certain local conditions in the abdomen which help to bring these states or conditions about. The most common pathology of mankind is found in the colon, mostly in the right half in small ecchymotic areas in the mucous membrane, and various degrees of cellular destruction in the cells of the intestinal tubules. While these pathologies are occasionally noted in the ileum, they more particularly are found in the right half of the colon and in the proximal portion of the transverse colon. They are the direct effects of toxic substances in the gut and usually run along symptomless in the individual.

Commonly (as I have drawn to reader's attention) there is also present degeneration in the ganglia of the Auerbach and Meissner plexuses and more or less destruction of the fibers which run to the ganglia or come from the various cells. When such a condition exists, intestinal stasis is commonly present and this increases the status of toxemia. The difficulties in the subject have been multiplied because under the term "intestinal stasis" have been included numerous conditions in the abdomen in which stasis was only functionally present. This has been brought about largely by the propaganda of Lane and quick and incomplete conclusions on x-ray methods of examination, operation often being performed on the basis of a stasis alone. These cases are designated as "intestinal stasis" but should be designated according to the pathologies found in which the intestinal stasis is an accompanying or secondary

condition. It is these facts that have brought about confusion and incomplete understanding of the subject of intestinal or colonic stases, which is unfortunate.

In not a few cases of intestinal toxemia, when the condition has existed for a long time and had been neglected, pathological change takes place in the mucous membrane. The first noticed is usually a swelling of the epithelium giving it a thickened glossy appearance and somewhat smoother than normal.



Fig. 7.—Pathology of Right Colon Due to Intestinal Toxemia. Cecum.

Showing normal mucous membrane and caput cecum above ileocecal valve, chronic inflammation of the ileocecal valve (which was firm and closed when tissue was removed but is shown relaxed because the specimen was one day old when photographed), areas of hemorrhage and ulceration in the cecum beyond the valve, the mucous membrane atrophic and the gut wall thinned.

In the course of time a lytic necrosis added to by round cell infiltration in the mucosa causes an absorption of the elements of the mucous membrane, mainly the cellular, which is replaced by a stroma sometimes very homogeneous in character. Sections of such mucous membrane may show a complete absence of tubular cells, the mucous membrane presenting degenerative

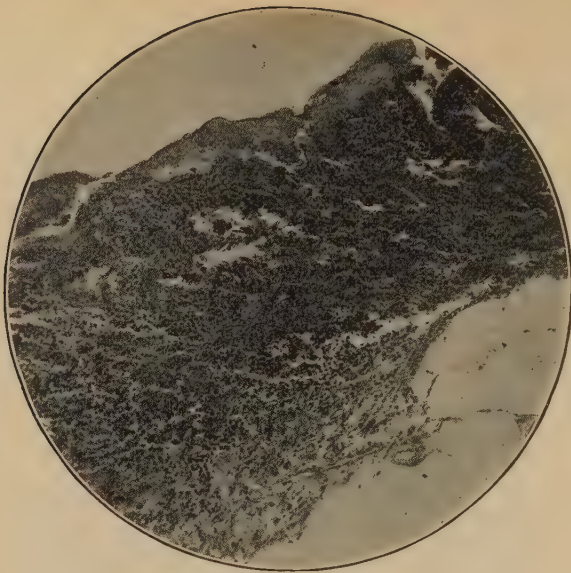


Fig. 8.—Degenerative Changes in the Mucosa in Intestinal Toxemia.

This specimen shows degeneration and necrosis in the mucosa, which is more particularly noticeable in the lymphadenoid tissue and glands, the latter not showing in this microphotograph, which shows a degeneration and necrotic area in the lymphadenoid tissue. There are no marked changes in the submucosa. $\times 145$.

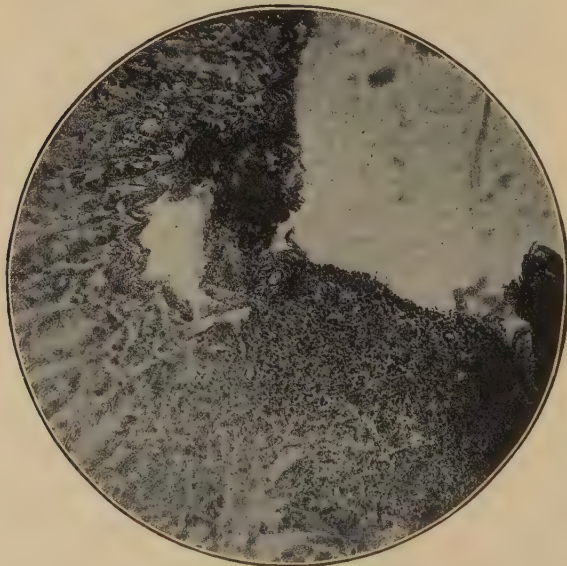


Fig. 9.—Degenerative Changes in the Mucosa in Intestinal Toxemia.

This specimen shows extensive degenerative and necrotic changes in the mucosa, among the glands and lymphadenoid tissue. Some of the glands are denuded of epithelium. There are some slight degenerative changes in the submucosa, but these are not nearly so marked as in the mucosa. Could detect no signs of hemorrhage. $\times 145$.

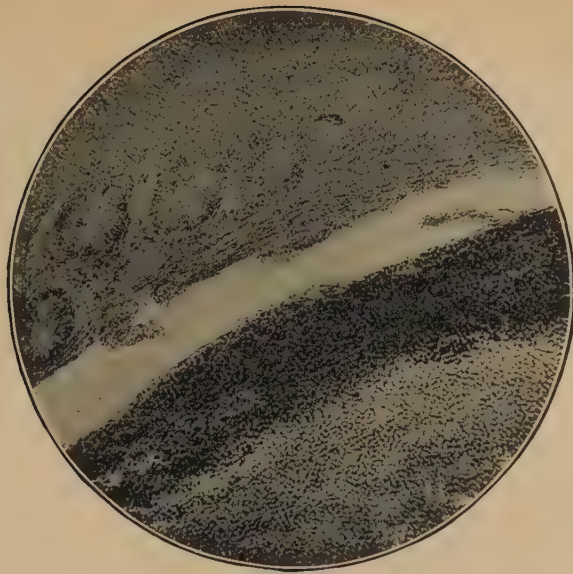


Fig. 10.—Degenerative Changes in the Mucosa in Intestinal Toxemia.

Like Figs. 8 and 9, this shows considerable degeneration and necrosis in the mucosa among the glands and lymph nodules, some of the former even showing desquamation of glandular tissue. There are also some slight degenerative changes in the submucosa (not shown in this picture). The specimen is truly atonic. $\times 145$.

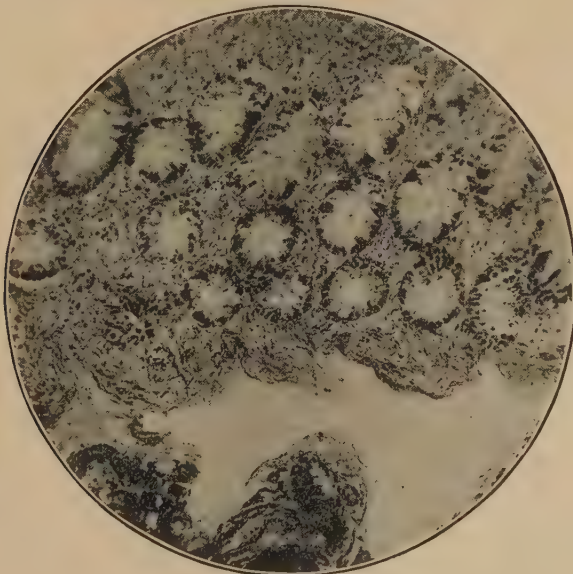


Fig. 11.—Degenerative Changes in the Mucosa in Intestinal Toxemia.

This specimen is more nearly normal than any of those shown (Figs. 8, 9, 10), but in places it still shows degeneration and necrosis in the mucosa among the glands and lymphoid tissue. All such foci in the stained specimen are of much paler color than in the normal areas. $\times 145$.

and abnormal material noticeable mainly in the lymphadenoid tissue and glands. This degeneration and necrosis extends, causing a desquamation of the cellular tissue, finally the total destruction of the glandular tissue, and perhaps some slight degenerative changes in the submucosa.

Viewing such mucous membrane, it looks dry and thin, and here and there are hemorrhagic areas, sometimes with



Fig. 12.—Normal Ganglion of Auerbach Plexus. (*Ehrlich* method.)

small ulceration. It is my experience these conditions are not met with in the caput of the colon, but correspond to the course of food through the gut, the ileocecal valve area being tremendously injected or congested, but showing nothing more than a simple hyaline swelling in the superficial layers of the mucous membrane. As the condition continues and the pathologic changes in ganglionic cells of the Auerbach and Meissner plexuses occur, the ganglionic cells become degenerated at the outer aspects, a fine granular degeneration commonly extending almost to the center of the cell.

This degeneration is in the protoplasm and neurons of the cell and seriously interferes with its function, which function is further interfered with by the afferent and efferent branches going to and from the main and remote collections of ganglionic cells; these wither up in some instances, in others they become absolutely detached, absorbed, and usually in all instances the total number is tremendously diminished.

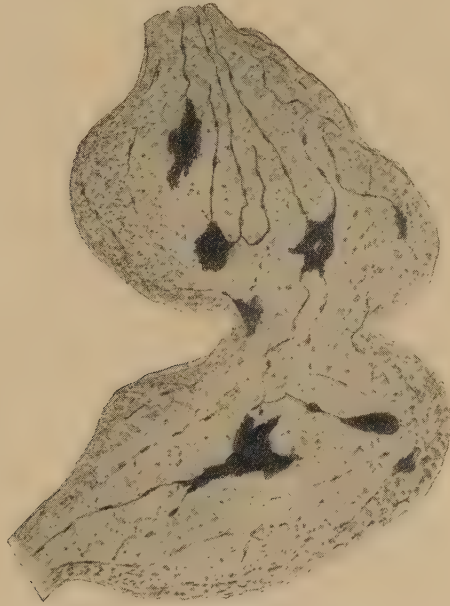


Fig. 13.—Ganglion from Auerbach Plexus in a Case of Chronic Toxemia.

Showing shrunken ganglia and fine granular degeneration of the protoplasm.

Manifestly, such a condition interfering definitely with the innervation of the muscular fibers, mainly the muscularis mucosæ and circular, produces what is called "intestinal stasis." This pathology may be secondary to conditions such as bands, kinks, ptosis and others, although it is rarely possible to state definitely that the condition is secondary even though these anatomical defects are noted. For commonly these anatomical defects are themselves secondary to an original chronic

intestinal toxemia which has not been discovered, as no symptoms have arisen until definite stasis has existed, when they are erroneously ascribed to those of stasis entirely. If the medical profession will keep this understanding of intestinal stasis in mind there will be no confusion as to how the term "intestinal stasis" should be understood and used.

These conditions also by seriously interfering with the innervation and the anatomy of the mucous membrane in digestive ability and secretion, bring about certain enhancing effects on the intestinal toxemia that is present. The gut undertakes to protect itself, commonly causing an increase in mucus discharge, this being usually seen in the stools, a state which is erroneously designated as chronic colitis, or colitis, or one judged as neurological. The first is a symptom and not an entity, and the second a fourth stage consequence. The course of development here is first an intestinal toxemia, second gut pathology, third increased mucus, and fourth neurological upset. As stated before, these pathologies are exceedingly common, perhaps existent in at least one-half the middle-aged adults, and insofar as so-called spastic constipation or atrophic constipation or colitis are concerned they all are not understood by the profession nor handled intelligently in the light of the main etiological factors.

In numerous instances of careful dissection work, and the study of minute anatomy of the abdomen, it has been proven endless times that this pathology in the bowel is capable of being extended, insofar as the sympathetic nervous system is concerned, along the course of the fibers and even to the central cells of the thoracic and abdominal course of the ganglionic sympathetic centers. States like incompetency of the ileocecal valve, colitis, sigmoiditis, pericolonic adhesions, caput puddling, so-called intestinal acidosis, dilatations in various portions of the gut (these even as remote as in the stomach) are consequential conditions originally caused by intestinal toxemia, and to repeat, when these minute pathologies are present they invariably enhance the status of toxemia within the intestinal canal.

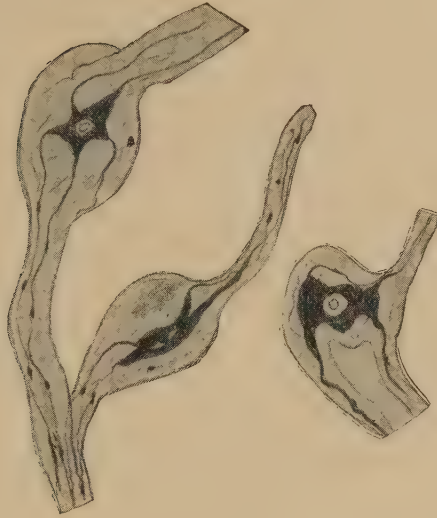


Fig. 14.—Cells of the Meissner Plexus. Normal Human Being.
(*Ehrlich* method.)

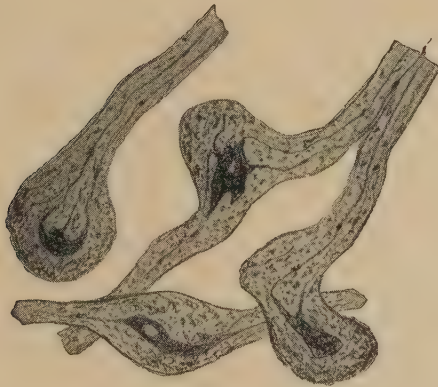


Fig. 15.—Cells of Meissner Plexus in a Case of Chronic
Intestinal Toxemia.

Cells markedly shrunken and degenerated. Poor staining of nucleus.

The above represents some of the conditions that ensue which have to do with symptom and disorder production. They are largely brought about by toxins operating in various ways but initially originating in the intestine. The most important of these are the toxins produced by the organisms themselves, and chemistry and biology will never advance to the perfection of their isolation, examination and estimation from the blood, tissues or the eliminations from the body, not even in the feces, except indirectly by bacterial methods. Regarding these the future will show that they are the most potent of all poisons known to warm-blooded animals, very trivial doses being lethal.

What is still more confusing in the intestinal states in the way of estimations will be the combinations of toxins—and they are almost the rule in these disorders—and again these combinations acted upon by the body tissues with the production of new toxic entities. In addition, are the food-bacterial poisons produced in the bowel, practically all of which are resorbable, whether in soluble or gaseous forms, into the body, these also changing into new toxic substances when coming in contact with the body tissues.

Important, too, are the conditions in abilities or deficiencies of the conjugating or combining substances, the factors of deficient tissue metabolism, the oxidizing ferments and factors, and finally those of elimination from the body. All of these represent entities, complexes, mixture products and results, regarding which analytical medicine has to go a long way in development of biological medicine before they are within chemical grasp or complete understanding. It is today enough to know that these states exist, and that stools collected and examined under proper conditions and by proper biological methods put the subject within the grasp of organism identifications. When these have been made, because of the ever-varying pictures and combinations met with, final conclusions are an art in fitting the significances, and a matter of observation and experience. But in these methods of stool examination there will always remain one great asset which future developments of the subject will not obviate or modify, and

that is, whatever the means of blood, urine and other types of analyses, these can be only most indirect, while biologically that of stools will be direct and thus more accurate.

The above represents the general biologic, the environmental, the contributing and toxic factors of intestinal toxemia. The actual and true etiological factor is represented in the bacteriology of the intestinal canal, and it is only when the subject of intestinal toxemia is studied and viewed from this bacteriologic standpoint that it can be understood and that methods for its handling (instead of being stupid as they are today) can be scientific and right. It is unfortunate that the commonest disease and disorder of the human being, prevalent throughout the world in endless numbers of cases, has turned out to be the most difficult of diagnosis of any and all disorders, and one in which only bacteriological procedures can be engaged in for its discernment. And the therapy, being entirely a biologic and bacteriologic affair, is also difficult.

Together with the many factors that are mentioned in various parts of this volume which have to do with the production of a toxic individual, there is one angle of this subject that should be mentioned for understanding of it. In the intestinal canal toxemias are not due, in the majority of instances, to one or more organisms working upon the food pabulum at only one level of the gut. As a rule the process is gradient. Starting in relatively simple ways at a high level, this is increased by biological additions as the bowel contents descend. On the way down, process is added upon process, one bacterium or set of bacteria preparing the way for the subsequent ones.

Although in the higher levels not much toxic material is resorbed into the body, in the lower levels of the ileum and colon the finished toxic products are most readily resorbed. When foods are in the small intestine, the business of this sector of the bowel is mostly digestive, to maintain heat, nutrition, body functions, etc. This has largely been accomplished when the ileo-cecal region has been reached.

Beginning in the lower ileum, function starts to prepare the contents for elimination—resorption, of course, still continuing. As portion after portion is sent into the cecum, that

in the cecum is churned about by forward, retrograde, circumferential and shortening movements. At this time two distinct results take place: a most active resorption to take up the remainder of the food substances, fluids and the enzymes, and the second, the beginning of inspissation, which inspissation phenomenon continues along the rest of the colon. It is during the time of final resorption and inspissation that the finished toxic products go into the system, but the real biologic factors engaged in the production of these toxic substances began and built up these toxins in the higher levels of the small intestine. It therefore must be evident that to break up this sequential chain of events knowledge of all the bacteria present and all biological matters pertaining to them be known, as well as those that pertain to immunity production or other forms of their biologic control in treatment.

CHAPTER IX.

IMPORTANCE OF SEPARATING THE SECONDARY FROM THE PRIMARY TOXEMIAS.

Propaganda has been conducted in medicine for the past several years drawing attention to the importance of complete examination of patients. Years ago a well-grounded clinician could by the exercise of his faculties, backed up by past clinical experience, equip himself satisfactorily for practice. This in past years when the knowledge of medicine and disorders was limited, and when clinical experience and personal attendance comprised about all that was known, made the service satisfactory all around. But with the advent and development of surgery, specialism, the x-ray, laboratory procedures, bacteriology and especially biological therapeutics, and the familiar use of the specialized methods and apparatus of examination, the days of depending upon one's clinical abilities solely, with a clinical thermometer and a stethoscope and a few minutes' time to make a diagnosis, have largely gone. Still there are many practitioners and even some specialists who render no better service than this today. So common is this still, that one may rightfully wonder if the problems and difficulties which medical men are complaining about today are not more due to the shortcoming in ability and the lack of careful work than any other reason.

In no line of work in medicine is this situation more lamentably present than in the diagnoses and treatments of the intestinal toxemias. The average general practitioner burdened with work usually makes the diagnosis in a more or less tentative way. Sometimes he is correct and more times not. If correct, he generally is devoid of analytical ability to diagnose this in detail and depends upon a makeshift diet, medications which never cure, and perhaps colonic irrigations which do not either. Yet, for this general practitioner I have the kindest feeling because he at least makes a sort of assumptive diagnosis, which is more than can be said of the majority

of specialists. The latter usually neglect the condition altogether.

Among the higher grades of general practitioners, some of the internists, and others, efforts are made to accomplish more or less detail in the diagnosis by having the feces, urine and perhaps the blood, examined by some laboratory and significance placed upon the examinations made. In these past thirty years I have seen hundreds of such reports and I have not seen one yet that is of value, and this includes the reports from sanatoria which advertise special ability and equipment in the handling of this subject, and those from the laboratories of many of our best-known hospitals and the most active of the private laboratories in the large cities. With practically all the men whose work is that of handling digestive disturbances, quite as lamentable a situation exists.

Of late years though (not infrequently from those who have talked the most definitely and written the most trenchantly against there being any such state as intestinal toxemia and have belittled the importance of the biological side of this subject), one now hears: "Dr. Short says that I have four types of streptococci in my stools," or Dr. Long says, "Mr. Past died of a germ in his intestine which was a very bad one," and statements like these. Manifestly, many of the patients of these men have had their stools examined, and to the majority of people this is taken as sufficient to include anything untoward that may be found present. On questioning here, however, it has always been evident to me that the examinations in kinds of stools, how obtained, and the short time before the biological diagnosis is given, proves conclusively that if bacteriological examinations were made, they generally could be only of the superficial and worthless kind.

It is perfectly practical for a laboratory worker, after a few years spent in study and work on the examination of stools, to become proficient and to give to the medical attendant a biological diagnosis which he could interpret in connection with the case at hand. But it is required, before good work in treatment be started, that the medical attendant be quite as familiar with all of the laboratory procedures engaged in, how

the biologic conclusions were arrived at, and be even more critical and familiar with the work of the laboratory than the laboratory worker himself. If the biologic types of treatment which I am presenting are engaged in, the closest checking, rechecking, indices for shifts and modifications are so constantly required that about all the laboratory worker would be useful for would be to do the technical work, and the physician himself be also a bacteriologist as well as therapist. Should there be a laboratory technician in the work, this unquestionably requires that the laboratory be at the right hand of the physician every moment and that the physician's knowledge of the laboratory and the subject be extensive and that he himself put in many hours of the day in laboratory work.

There is no other way by which justice can be done to this work. One may make short shrift of it and occasionally do well here and there, but in only the average run of toxemias this will fail in more than half of the cases, and in the unusual types (and they are many) the failures would be complete. There is no doubt that, in the commonest of illnesses that the human being has, for proper treatment, none other is so difficult of study and handling, consumes so much time, is so expensive to conduct, and requires so much experience, knowledge and constant scientific observation.

DISTINGUISHING PRIMARY AND SECONDARY FORMS OF INTESTINAL TOXEMIA.

There is no one fact of greater importance in the handling of intestinal toxemia than the separation of the secondary from the primary form. Not only is this important from the standpoint of the scientific and the best type of medical management, but it is also of economic importance in behalf of the patient. To employ biologic types of treatment where the toxemia is secondary or only symptomatic of some other condition is manifestly unfair to the patient, and unfair to the scientific side of intestinal toxemia and medicine.

If in the handling of such a toxemia, only dietetics, medicinal or irrigation methods are employed, not much criticism

could be made, but to handle a secondary toxemia in the biologic ways brought out in this volume and to pay little or no attention to the causative condition, should not be the policy of a conscientious physician. Up to the present, however, so little has been known of diagnostic methods and biological treatments of intestinal toxemia, all instances of secondary and primary being treated in similar ways, that practically no harm has been done. But those who undertake to handle the subject as herein suggested, are as physicians honor bound not to treat a secondary toxemia in the biologic way one would use in the primary forms. One of the main reasons I have not written on this subject before was because of the fear that this would happen either through ignorance, deficiency of knowledge of the conditions of the patient from incomplete examinations, poor clinical interpretation or abilities of the attendant, or downright unpardonable commercial exploitation.

In judging whether one deals fundamentally with a secondary or a primary toxemia, all that is represented in the faculties and diagnostic abilities of a good internist as well as a gastro-enterologist is required. A sufficiently complete examination of the patient is required in all that such represents. This comprises data in all matters pertaining to the illnesses that the human being is heir to. The advice is given not to make a diagnosis of basic types of intestinal toxemia until all other disorders and diseases have been ruled out—to think of it the last of all. Running back in the case histories of the last five years of patients who in the history, physical examination, and the laboratory have suggested the presence of an intestinal toxemia, or who have come in for treatment in this connection, the following diagnoses have been fundamentally made. In the presence of a distinct disease this takes precedence over an intestinal condition, even when a primary toxemia can be diagnosed. In not a few of the following the presence of a chronic intestinal toxemia was proven, but the handling of the case (with exceptions) was only that of the major clinical condition. In well-established or advanced disease, restitution of pathology cannot take place from intestinal handling.

- Nephritis, advanced.
Gastric ulcer.
Duodenal ulcer.
Emphysema.
Aneurism, thoracic.
Hysteria, fundamental type.
Muscular dystrophy.
Gastric cancer.
Rectal cancer.
Colon cancer.
Gall-bladder cancer.
Ptosis, congenital.
Ptosis, Landau.
Chronic bronchitis, other causes.
Myxedema.
Diverticula, Meckel's.
Malaria.
Combined cord degeneration.
Pulmonary tuberculosis.
Chronic infective tonsillitis.
Diverticula, duodenum.
Diverticula, colon.
Cecal bands, obstructive.
Simple hyperchlorhydria (dietetic).
Liver abscess.
Pancreatic cysts.
Mediastinal tumor.
Arteriosclerosis, advanced.
Chronic multiple neuritis.
Obesity, some types.
Nematodes.
Chronic lung abscess.
Pott's disease.
Senility.
Amyotrophic lateral sclerosis.
Banti's disease.
Cardiac insufficiency.
Tuberculous peritonitis.
Myocarditis, advanced.
Syphilis.
Intestinal obstruction.
Brain tumor.
Renal stones.
Epilepsy, fundamental types.
Paralysis agitans.
Acromegaly.
Spinal cord tumor.
Endocarditis.
Sapremia.
Cirrhosis of liver, advanced.
Paresis.
Leukemia.
Hydronephrosis.
Salpingitis.
Amebic dysentery.
Renal tuberculosis.
Chronic esophagitis.
Locomotor ataxia.
Gall-stones, with symptoms.
Chronic pancreatitis.
Bronchiectasis.
Pericarditis.
Splenic anemia.
Pernicious anemia, too advanced.
Raynaud's disease.
Lead poisoning.
Sacroiliac disease, trauma.
Angina pectoris, plainly evident.
Diabetes mellitus.
Serous meningitis.
Dietl's crisis.
Fatty heart.
Bone abscess.
Apoplexy.
Diabetes insipidus.
Echinococcus cysts.
Hour-glass stomach.
Paget's disease.
Chronic skin disorders, some.
Vincent's infection.
Depressive mania.
Endocervicitis.
Uterine fibroids.
Enlarged prostate.
Cystic kidneys.
Sinusitis, surgical types.
Neuralgia, from other causes.
Sciatica, from other causes.
Psychasthenia, from other causes.
Neuritis, from other causes.
Aneurism, abdominal.

In addition to the above when the following facts were evident in the habits and conditions of the individual they were commonly taken as reasons for the complaints and judged as causative or for the time being as secondary cases. Not a few of such, however, finally prove to be instances of primary toxemias.

Excessive tea drinking.	Too much working.
Excessive coffee drinking.	Too much worrying.
Excessive alcohol drinking.	Too much fussing.
Improper eating habits.	Too much anxiety.
Tobacco poisoning.	Too much grief.
Over-eating.	Too stout.
Carelessness in bowel movements.	Too thin.
Ignorance in bowel movements.	Advanced old age.
False modesty in bowel movements.	Excessive use of purgatives.
Too little exercise.	Excessive use of anilines.
Too little fresh air.	Sexual excesses.
Too little sleep.	

Going over still more records no doubt other fundamental conditions would be found recorded, but enough are presented to prove that these diseases, disorders, and general factors, when present, are not to be judged or handled primarily in intestinal ways. In not a few of the above cases the diseases recorded were not markedly advanced, all of the patients being perfectly able to get about and come to the office of any physician, and being able to travel long distances to do so. It therefore is most essential that all patients be studied completely in advance of any thoughts being given to the intestines as a cause for their complaints.

When any disease, disorder or set of conditions cannot be found to explain the presence of the patient before you, and there is reason in the history, physical examination and the findings on examination of fresh urines, the possibility of an intestinal toxemia may be looked for. If, as a result of the examinations such be found, one is safely (but not always) in the realm of a suggestive diagnosis of some primary form of intestinal toxemia. I say not always, because, firstly, some diseases and disorders may exist and because of paucity of symptoms not be diagnosable, and secondly, the further laboratory examinations must prove an intestinal toxemia in

definite ways. If it does not, I would advise that this diagnosis be not made, no matter how suggestive the history and physical examination may be. In such situations I have found that, on going over the history and examinations again as if it were the first time, facts are uncovered which reclassify the case usually as one of some type of disorder easily to be benefited by simple or ordinary means, or hopelessly incurable by treatments, or those in which only symptomatic benefit can be accomplished.

CHAPTER X.

THE INTESTINAL ORGANISMS.

The lower end of the small intestine and the large intestine of man is a veritable culture tube of innumerable organisms, struggling constantly to gain supremacy over one another. It has been variously estimated that feces contain from one-third to one-half solid weight of bacteria, or a daily output of about 125 billion. Against these the stomach possesses a slight antiseptic property, owing to the acidity of the gastric juice, but its function as a germ-destroying organ is practically negligible. The usually small bacterial content of the duodenum and jejunum may be explained by dilution with large amounts of germ-free intestinal juice, which in itself has been shown not to be bactericidal.

Cultural methods known today suggest that most of the organisms voided are non-viable. This is only partly true and nowhere near 99 per cent., as most observers have believed. The death rate of these bacteria found in stools is high, due to cold and old specimens having been examined, a disintegration of some in their own juices (autolysis), and poor and deficient types of cultural methods having been employed.

If the specimens examined are obtained as the author suggests for proper biological examination, cultural procedures begun promptly with the warm specimen (within thirty minutes and kept warm until begun) and anaërobic procedures followed as well as the aërobic ones, the rate of viability may be as high as 70 per cent. in successive specimens. In constipated stools which are cold and examined some time after voiding, the percentage of dead forms is commonly as high as 99 per cent.

OCCURRENCE IN THE INTESTINAL TRACT.

The digestive tract of normal infants is sterile at birth, adventitious organisms making their entrance through the mouth and anus soon thereafter, which give way to certain definite

types as soon as the infant is placed on an all-milk diet. Tissier made the observation that the predominating organism of the nursling's flora is a Gram-positive bacillus, *Lactobacillus bifidus* (Tissier), which persists as the outstanding organism as long as mother's milk constitutes the entire food. Before his time, Escherich described for the first time the organism of the *B. coli* class (old term used), which is so characteristically an intestinal bacterium.

About the time that Tissier drew attention to the *B. bifidus*, Moro drew attention to an organism closely related to it in many respects and prominent in nursling's stools, this organism today corresponding to the *B. acidophilus*. As the child grows older and the diet becomes more varied the flora changes from the simple to one that is more complex, and that of the ordinary adult usually contains but few of the aciduric organisms (*B. bifidus* and *B. acidophilus*), the stool usually containing more predominantly those of the *Escherichia coli* grouping and other genus types.

Of yeasts and molds there are a large number found in the intestines, and there is some doubt that they can multiply in the canal, although ordinary yeasts may resist the antagonistic forces of the digestive tract and pass through more or less unharmed. The molds are passed from the intestines chiefly in the spore stage.

CLASSIFICATION.

Bacteriological literature is replete with classifications of bacteria of every sort, and to attempt to analyze these here is about as hopeless as a new classification would be useful. Various workers have classified them on aërobic and anaërobic bases and some on cultural characteristics, especially their behavior in various media. Classification has been attempted on whether they are of animal or vegetable origin, likewise other classifications have been advanced on the basis of their morphology, their staining characteristics, etc. The committee of the Society of American Bacteriologists stated in 1917 that "the name need not be appropriate, it need only be stable."

There is no more chaotic status in medicine than the present methods of bacterial classification, and rather than confuse the reader with a subject already difficult, the new names of organisms are used, and, when possible, their old names are mentioned with the new nomenclature as synonymous terms.

Various microorganisms known to bacteriology are classified variously by different investigators under the general term of Schizomycetes. It is quite out of place in this volume to present all of the detail connected with bacteriology in its general and systematic knowledge. In this connection the reader is referred more particularly to the works of Buchanan, Chester, Castellani and Chalmers, Bergey, Ford, Hiss and Zinsser, and Kendall. For the more complete study of microorganisms the above-mentioned books should be employed as guides. In this volume general classifications are made under the pathogenes, saprophytes and zymogenes with relative matter pertaining to the non-pathogenic forms. In this, the new classifications of Bergey are more particularly employed, although at times these are departed from when they do not answer to sufficient purpose.

In connection with the different organisms mentioned are notes regarding incidence, associations and suggestions of biologic treatments to be employed. In an ever-shifting subject as one studies case after case only suggestions can be mentioned. More than this, in the same case as treatment progresses, new significances as to the original findings are constantly occurring, these changing the treatment again. Whatever the original bacterial diagnosis may be, complete re-examinations every two weeks are required because these prove or disprove the accuracy of the original biologic diagnosis and give additional data and suggestions. The average case under treatment usually requires the performance of fifteen complete biological examinations of stools for thirty weeks of treatment, and then one a month after the treatments have ceased for a period of six months.

Intestinal Pathogenic Organisms.

<i>Terms Used.</i>	<i>Terms Discarded.</i>
<i>Alcaligenes fecalis.</i>	<i>Bacillus fæcalis alcaligenes</i> Petruschky.
<i>Bacillus cavicidus</i> Brieger.	<i>Bacillus cavicidus</i> Brieger, 1884.
<i>Bacillus furcosus.</i>	<i>Bacillus furcosus</i> Veillon and Zuber.
<i>Bacillus fusiformis.</i>	<i>Bacillus fusiformis</i> Gottheil.
<i>Clostridium edematis maligni.</i>	<i>Vibrio septique</i> Pasteur, 1877. <i>Bacillus sporogenes.</i> Synonyms: <i>Bacillus</i> of Ghon and Sachs; <i>Bacillus</i> III of von Hibler.
<i>Clostridium putrificum.</i>	<i>Bacillus putrificum.</i> <i>Bacillus putrificus coli.</i>
<i>Clostridium tetani.</i>	<i>Bacillus tetani</i> Nicolaier.
<i>Diplococcus pneumoniae.</i>	<i>Microbe septicémique du saliva</i> Pasteur, 1881.
	<i>Micrococcus Pasteuri</i> Sternberg, 1881.
	<i>Pneumococcus Friedländer,</i> 1882.
	<i>Lancet-shaped micrococcus</i> Talamon, 1883.
	<i>Pneumococcus Fränkel,</i> 1886.
	<i>Bacillus septicus sputigenus</i> Flügge, 1886.
	<i>Bacillus salivarius septicus</i> Biondi, 1886.
	<i>Streptococcus lanceolatus Pasteuri</i> Gamaleia, 1888.
	<i>Bacterium pneumoniae</i> Migula, 1900.
	<i>Pneumococcus.</i>
<i>Eberthella belfastiensis.</i>	<i>Bacillus belfastiensis</i> Wilson, 1908.
<i>Eberthella dispar.</i>	<i>Bacillus dispar</i> Andrews, 1918.
<i>Eberthella douglasi.</i>	<i>Bacillus douglasi</i> Castellani and Chalmers, 1918.
<i>Eberthella dysenteriae.</i>	<i>Bacillus dysenteriae</i> Shiga-Kruse.
	<i>Bacillus dysenteriae</i> Y of Hiss and Russell.
<i>Eberthella kandiensis.</i>	<i>Bacillus kandiensis</i> Castellani.
<i>Eberthella lunavensis.</i>	<i>Bacillus lunavensis</i> Castellani.
<i>Eberthella metadysenteriae,</i> Variety A.	<i>Bacillus metadysentericus</i> Castellani, 1904, Variety A.
<i>Eberthella metadysenteriae,</i> Variety B.	<i>Bacillus metadysentericus</i> Castellani, 1904, Variety B.
<i>Eberthella metadysenteriae,</i> Variety C.	<i>Bacillus metadysentericus</i> Castellani, 1904, Variety C.
<i>Eberthella metadysenteriae,</i> Variety D.	<i>Bacillus metadysentericus</i> Castellani, 1904, Variety D.
<i>Eberthella minutissima.</i>	<i>Bacterium minutissimum</i> Kruse, 1886
<i>Eberthella paradysenteriae.</i>	<i>Bacillus dysenteriae</i> Flexner, 1900.
<i>Eberthella pritznitzii.</i>	<i>Bacillus pritznitzii</i> Castellani.
<i>Eberthella pyogenes.</i>	<i>Bacillus pyogenes fetidus</i> Passet, 1885.
<i>Eberthella tangallensis.</i>	<i>Bacillus tangallensis</i> Castellani.
<i>Eberthella typhi.</i>	<i>Bacillus der abdominal Typhus</i> Eberth, 1880.
	<i>Typhus bacillen</i> Gaffky, 1884.
	<i>Bacillus typhosus</i> Zopf, 1885.
	<i>Bacillus typhi</i> Cohn, 1886.
	<i>Bacillus typhi abdominalis</i> Flügge, 1886.

Intestinal Pathogenic Organisms (*Continued*).

<i>Terms Used.</i>	<i>Terms Discarded.</i>
	<i>Vibrio typhosus</i> Trevisan, 1889.
	<i>Eberthus typhosus</i> Castellani and Chalmers, 1919.
<i>Erythrobacillus prodigiosus.</i>	<i>Serratia marcescens</i> Bizio, 1823.
	<i>Zaogalactina imetrofa</i> Sette, 1824.
	<i>Protococcus imetrophus</i> Meneghini, 1838.
	<i>Monas prodigiosa</i> Ehrenberg, 1839.
	<i>Palmella prodigiosa</i> Montague, 1853.
	<i>Micraloa prodigiosa</i> Zanardini, 1863.
	<i>Micrococcus prodigiosus</i> Cohn, 1872.
	<i>Bacteridium prodigiosum</i> Schröter, 1872.
	<i>Bacillus prodigiosus</i> Flügge, 1886.
	<i>Bacillus imetrophus</i> Trevisan, 1887.
	<i>Bacillus marcescens</i> De Toni and Trevisan, 1889.
	<i>Bacterium prodigiosum</i> Lehmann and Neumann, 1896.
	<i>Liquidobacterium prodigiosum</i> Orla-Jensen, 1909.
<i>Escherichia carolinus.</i>	<i>Bacillus carolinus</i> Castellani.
<i>Escherichia coagulans.</i>	<i>Bacillus coagulans</i> Castellani.
<i>Escherichia coli.</i>	<i>Bacterium coli commune</i> Escherich, 1886.
	<i>Bacillus coli communis</i> (Escherich) Sternberg, 1892.
	<i>Bacillus coli verus.</i>
	<i>Bacterium coli</i> (Escherich) Lehmann and Neumann, 1896.
	<i>Bacillus coli</i> (Escherich) Migula, 1900.
	<i>Bacillus coli similis</i> Sternberg.
	<i>Bacillus</i> of Jefferies.
	<i>Bacillus coli immobilis</i> Germano and Maurea, 1892.
	<i>Bacillus coli immobilis</i> Kruse, 1896.
	<i>Bacillus coli mobilis</i> Messea, 1890.
	<i>Bacillus monadiformis</i> Kruse, 1896.
	<i>Bacillus putrificus coli</i> Bienstock.
<i>Escherichia communior.</i>	<i>Bacillus coli communior</i> Durham, 1900.
<i>Escherichia grünthali.</i>	<i>Bacterium grünthali</i> Fisher, 1902.
<i>Escherichia leporis.</i>	<i>Bacillus leporis lethalis</i> Gibier and Sternberg, 1890.
<i>Escherichia paragrünthali.</i>	<i>Bacillus paragrünthali</i> Castellani and Chalmers, 1919.
<i>Proteus valeriæ.</i>	<i>Bacillus valeriei</i> Boycott, 1906.
<i>Proteus vulgaris.</i>	<i>Bacillus proteus vulgaris</i> Hauser.
<i>Pseudomonas æruginosa.</i>	<i>Bacterium æruginosum</i> Schröter, 1872.
	<i>Bacillus pyocyaneus</i> Gessard, 1882.

Intestinal Pathogenic Organisms (*Continued*).

<i>Terms Used.</i>	<i>Terms Discarded.</i>
<i>Pseudomonas non-liquefaciens.</i>	<i>Pseudomonas aeruginosa</i> Migula, 1896.
<i>Salmonella aertrycke.</i>	<i>Bacterium pyocyaneum</i> Lehmann u. Neumann, 1896.
<i>Salmonella enteritidis.</i>	<i>Bacillus fluorescens non-liquefaciens.</i>
<i>Salmonella mobilis.</i>	Eisenberg, 1891. <i>Bacillus</i> of Lesage.
	<i>Bacillus aertrycke</i> De Nobele, 1889.
	<i>Bacillus enteritidis</i> Gaertner.
	<i>Bacillus pseudo-asiaticus mobilis</i> Castellani, 1915.
<i>Salmonella negombensis.</i>	<i>Bacillus negombensis</i> Castellani, 1910.
<i>Salmonella para-aertrycke.</i>	<i>Bacillus para-aertrycke</i> Castellani, 1914.
<i>Salmonella para-asiatica.</i>	<i>Bacillus para-asiatica</i> Castellani, 1916.
<i>Salmonella paradiffluens.</i>	<i>Bacillus paradiffluens</i> Castellani.
<i>Salmonella pseudo-asiatica.</i>	<i>Bacillus pseudo-asiaticus</i> Castellani, 1913.
<i>Salmonella pseudomorgani.</i>	<i>Bacillus pseudomorgani</i> Castellani.
<i>Salmonella pseudo-wesenbergi.</i>	<i>Bacillus pseudo-wesenbergi</i> Castellani, 1918.
<i>Salmonella Schotmülleri A.</i>	<i>Bacillus paratyphosus A</i> Schotmüller, Brion Kayser.
<i>Salmonella Schotmülleri B.</i>	<i>Bacillus paratyphosus B</i> Schotmüller.
<i>Salmonella willegodai.</i>	<i>Bacillus willegodai</i> Castellani.
<i>Staphylococcus albus.</i>	<i>Micrococcus pyogenes albus</i> Rosenbach, in association with <i>Staphylococcus aureus</i> .
	<i>Micrococcus pyogenes</i> (Rosenbach) migula.
<i>Vibrio comma.</i>	<i>Komma bacillus</i> Koch, 1884.
	<i>Spirillum cholerae asiaticæ</i> , <i>Comma bacillus</i> .
<i>Vibrio ghinda.</i>	<i>Vibrio ghinda</i> , Pfeiffer.
<i>Vibrio maszauah.</i>	<i>Vibrio maszauah</i> Pasquale.
<i>Streptococcus anginosus.</i>	<i>Streptococcus anginosus</i> .
<i>Streptococcus equi.</i>	<i>Streptococcus equi</i> Schütz.
<i>Streptococcus equinus.</i>	<i>Streptococcus equinus</i> Andrews and Horder.
<i>Streptococcus faecalis.</i>	<i>Streptococcus fecalis</i> Andrews and Horder, 1906.
	<i>Enterococcus Thiercelin</i> , 1902.
	<i>Streptococcus faecium</i> Orla-Jensen.
	The lactic acid bacillus, 1919.
<i>Streptococcus hemolyticus.</i>	<i>Streptococcus hemolyticus</i> Holman.
<i>Streptococcus hemolyticus II.</i>	<i>Streptococcus hemolyticus II</i> Holman.
<i>Streptococcus hemolyticus III.</i>	<i>Streptococcus hemolyticus III</i> Holman.
<i>Streptococcus ignavus.</i>	<i>Streptococcus ignavus</i> Holman.
<i>Streptococcus infrequens.</i>	<i>Streptococcus infrequens</i> Holman.
<i>Streptococcus mitis.</i>	<i>Streptococcus mitis</i> Andrews and Horder.
<i>Streptococcus non-hemolyticus I.</i>	<i>Streptococcus non-hemolyticus I</i> Holman.

Intestinal Pathogenic Organisms (Continued).

<i>Terms Used.</i>	<i>Terms Discarded.</i>
<i>Streptococcus non-hemolyticus</i> II.	<i>Streptococcus non-hemolyticus</i> II Holman.
<i>Streptococcus non-hemolyticus</i> III.	<i>Streptococcus non-hemolyticus</i> III Holman.
<i>Streptococcus pyogenes</i>	<i>Streptococcus pyogenes</i> Rosenbach, 1884.
	<i>Streptococcus puerperalis</i> Arloing, 1884.
	<i>Streptococcus puerperalis</i> Fourneaux, 1912.
<i>Streptococcus salivarius.</i>	<i>Streptococcus salivarius</i> Andrews and Horder.
<i>Streptococcus subacidus.</i>	<i>Streptococcus subacidus.</i>

Intestinal Saprophytic Organisms.

<i>Achromobacter metadiffuens.</i>	<i>Bacillus metadiffuens</i> Castellani.
<i>Achromobacter paradysentericus.</i>	<i>Bacillus paradysentericus</i> Castellani, 1904.
<i>Achromobacter pseudocarinatus.</i>	<i>Bacillus pseudocarinatus</i> Castellani, 1917.
<i>Achromobacter tardus.</i>	<i>Bacillus tardus</i> Castellani, 1917.
<i>Alcaligenes Bookeri.</i>	<i>Bacillus A</i> of Booker Sternberg, 1892.
<i>Alcaligenes fæcaloides.</i>	<i>Bacillus fæcaloides</i> Castellani, 1915.
<i>Alcaligenes gintottensis.</i>	<i>Bacillus gintottensis</i> Castellani, 1910.
<i>Alcaligenes recti.</i>	<i>Bacterium recti</i> Ford, 1901.
<i>Alcaligenes zeylanicus.</i>	<i>Bacillus zeylanicus</i> Castellani, 1910.
<i>Bacillus centrosporus.</i>	<i>Bacillus centrosporus</i> Ford, 1916.
<i>Bacillus flexus.</i>	<i>Bacillus flexus</i> Batchelor, 1919.
<i>Bacillus fusus.</i>	<i>Bacillus fusus</i> Batchelor, 1919.
<i>Bacillus lautus.</i>	<i>Bacillus lautus</i> Batchelor, 1919.
<i>Bacillus megatherium.</i>	<i>Bacillus megatherium</i> De Barry, 1884.
	<i>Bacterium sessile</i> Migula, 1889.
	<i>Bacillus sessilis</i> Klein, 1889.
	<i>Bacterium brassicæ</i> (Pommer) Migula, 1886.
	<i>Bacterium pseudoanthracis</i> (Wahrlich) Migula, 1890.
	<i>Bacterium anthracoides</i> (Hueppe and Wood) Migula, 1896.
	<i>Bacterium flexile</i> Burchard, 1897, 1898.
	<i>Bacterium hirtum</i> Henrici, 1898.
<i>Bacillus mesentericus.</i>	<i>Bacillus mesentericus</i> Flügge, 1886.
<i>Bacillus mycoides.</i>	<i>Bacillus mycoides</i> Flügge, 1886.
	<i>Bacillus brassicæ</i> Pommer, 1886.
	<i>Bacillus ramosus</i> Frankland, 1889.
	<i>Bacterium casei</i> Adametz, 1889.
	<i>Wurzelbacillus</i> Fränkel, 1890.
	<i>Bacillus ramosus</i> Eisenberg, 1891.
	<i>Bacillus intricatus</i> Russell, 1892.

Intestinal Saprophytic Organisms (*Continued*).

<i>Terms Used.</i>	<i>Terms Discarded.</i>
<i>Bacillus tritus.</i>	<i>Bacillus radicosus</i> Zimmermann.
<i>Bacteroides acuminatus.</i>	<i>Bacillus implexus</i> Zimmermann.
<i>Bacteroides angulosus.</i>	<i>Bacillus tritus</i> Batchelor, 1919.
<i>Bacteroides bullosus.</i>	<i>Diplobacillus acuminatus</i> Distaso, 1917.
<i>Bacteroides cornutus.</i>	<i>Bacillus angulosus</i> Distaso, 1912.
<i>Bacteroides dimorphus.</i>	<i>Bacillus bullosus</i> Distaso, 1912.
<i>Bacteroides laevis.</i>	<i>Bacillus cornutus</i> Distaso, 1912.
<i>Bacteroides liquefaciens.</i>	<i>Bacillus dimorphus</i> Distaso, 1912.
<i>Bacteroides pseudoramosus.</i>	<i>Bacillus laevis</i> Distaso, 1912.
<i>Bacteroides tenuis.</i>	<i>Coccobacillus liquefaciens</i> Distaso, 1911.
<i>Bacteroides thetaiotaomicron.</i>	<i>Bacillus pseudoramosus</i> Distaso, 1912.
<i>Bacteroides variabilis.</i>	<i>Bacillus tenuis spatuliformis</i> Distaso, 1911.
<i>Bacteroides variegatus.</i>	<i>Bacillus thetaiotaomicron</i> Distaso, 1912.
<i>Eberthella alkalescens.</i>	<i>Bacillus variabilis</i> Distaso, 1912.
<i>Eberthella ambigua.</i>	<i>Bacillus variegatus</i> Distaso, 1912.
<i>Eberthella bienstockii.</i>	<i>Bacillus alkalescens</i> Andrews, 1916.
<i>Eberthella chylogena.</i>	<i>Bacillus ambiguus</i> Andrews, 1918.
<i>Eberthella dubia.</i>	<i>Bacterium bienstockii</i> Schröter, 1886.
<i>Eberthella enterica.</i>	<i>Bacterium chylogenum</i> Ford, 1901.
<i>Eberthella oxygenes.</i>	<i>Bacterium dubium</i> (Bleisch) Kruse, 1893.
<i>Eberthella oxyphila.</i>	<i>Bacterium entericum</i> Ford, 1901.
<i>Escherichia alcalescens.</i>	<i>Bacterium oxygenes</i> Ford, 1901.
<i>Escherichia coli.</i>	<i>Bacterium oxyphilum</i> Ford, 1901.
	<i>Bacterium alcalescens</i> Ford, 1901.
	<i>Bacillus coli similis</i> Sternberg.
	<i>Bacillus</i> of Jefferies.
<i>Escherichia gastrica.</i>	<i>Bacterium gastricum</i> Ford, 1901.
<i>Escherichia iliaca.</i>	<i>Bacterium iliaceum</i> Ford, 1919.
<i>Escherichia neapolitana.</i>	<i>Bacterium neapolitanum</i> Emmerich, 1885.
<i>Escherichia pseudocoloides,</i> Variety A.	<i>Bacillus pseudocoloides</i> , Variety A, Castellani, 1916.
<i>Escherichia pseudocoloides,</i> Variety B.	<i>Bacillus pseudocoloides</i> , Variety B, Castellani.
<i>Escherichia pseudodysenteriae.</i>	<i>Bacterium pseudodysentericum</i> Ford, 1901.
<i>Micrococcus ovalis.</i>	<i>Micrococcus ovalis</i> Escherich.
	<i>Diplococcus intestinalis</i> , Soor, Murath, Moro.
	Gram-positive cocci.
	<i>Enterococcus.</i>
<i>Pseudomonas capsulata.</i>	<i>Bacillus fluorescens capsulatus</i> Pottein, 1896.
<i>Serratia havaniensis.</i>	<i>Bacillus havaniensis</i> Sternberg, 1892.
<i>Vibrio proteus.</i>	<i>Komma Bacillus der Cholera Nostras</i> , Finkler and Prior, 1884.

Intestinal Zymogenic Organisms.

Terms Used.

Achromobacter ceylonensis A.
Achromobacter ceylonensis B.
Aërobacter aërogenes.

Aërobacter diffluens.
Clostridium alcaligenes.

Clostridium coagulans.
Clostridium dissolvens.
Clostridium filamentosum.

Clostridium filiformis.
Clostridium fissum.
Clostridium ovalaris.
Clostridium putrificum.
Clostridium regularis.
Clostridium saccharolyticum.

Clostridium welchii.

Clostridium zoëgleicum.

Terms Discarded.

Bacillus ceylonensis A Castellani, 1905.
Bacillus ceylonensis B Castellani, 1905.
Actinobacter dulait visqueaux Duclaux.
Actinobacter polymorphus Duclaux.
Bacterium lactis aërogenes Escherich.
Bacillus guillebeau c, v. Freudenreich.
Bacillus lactic aërogenes Harrison.
Bacillus aërogenes Kruse.
Bacillus f, Schardinger.
Bacillus No. 23, Gruber.
Bacterium aërogenes Holland.
Bacillus diffluens Castellani.
Bacillus anaërobicus alcaligenes Debono, 1912.
Bacillus sporogenes coagulans Debono, 1912.
Bacillus cellulose dissolvens Khouvine, 1923.
Bacillus putrificus filamentosus Distaso, 1911.
Bacillus regularis filiformis Debono, 1912.
Bacillus fissus Debono, 1912.
Bacillus putrificus ovalaris Debono, 1912.
Bacillus putrificus coli Bienstock.
Bacillus sporogenes regularis Distaso, 1911.
Bacillus sporogenes saccharolyticus Distaso, 1911.
Bacillus perfringens Veillon and Zuber, 1898.
Bacillus aërogenes capsulatus Welch and Nuttall, 1892.
Bacillus phlegmonis emphysema Tosi Fraenkel, 1902.
Bacillus saccharobutyricus immobilis Schattenfroh and Grassberger, 1900.
Bacillus enteritidis sporogenes Klein, 1915 proparte (see remarks).
Bacillus welchii Migula, 1900.
Bacillus cadaveris butyricus Bu Day, 1898.
Bacillus emphysematis vaginæ Lindenthal, 1897.
Bacillus of acute articular rheumatism Achalme, 1891.
This organism in strains has been divided into types according to their action on carbohydrates. { Type I, Simonds
Type II, Simonds
Type III, Simonds
Type IV, Simonds
Bacillus sporogenes zoëgleicus Distaso, 1911.

Intestinal Zymogenic Organisms (*Continued*).

<i>Terms Used.</i>	<i>Terms Discarded.</i>
<i>Eberthella bentottensis.</i>	<i>Bacillus bentottensis</i> Castellani, 1905.
<i>Eberthella talavensis.</i>	<i>Bacillus talavensis</i> Castellani, 1909.
<i>Escherichia acidi-lactici.</i>	<i>Bacterium acidi-lactici</i> Hueppe, 1884.
<i>Escherichia coli.</i>	<i>Bacillus coli immobilis</i> Germano and Maurea, 1892.
	<i>Bacillus coli immobilis</i> Kruse, 1896.
	<i>Bacillus coli mobilis</i> Messea, 1890.
	<i>Bacillus monadiformis</i> Kruse, 1896.
	<i>Bacillus putrificus coli</i> Bienstock.
<i>Escherichia colitropicalis.</i>	<i>Bacillus colitropicalis</i> Castellani, 1907.
<i>Escherichia coloides,</i> Variety A.	<i>Bacillus coloides</i> , Variety A, Castellani.
<i>Escherichia coloides,</i> Variety B.	<i>Bacillus coloides</i> , Variety B, Castellani.
<i>Escherichia galactophila.</i>	<i>Bacterium galactophilum</i> Ford, 1901.
<i>Escherichia khartoumensis.</i>	<i>Bacillus khartoumensis</i> Chalmers and MacDonald, 1915.
<i>Escherichia metacoli.</i>	<i>Bacillus metacoli</i> Castellani, 1915.
<i>Escherichia metacoloides.</i>	<i>Bacillus metacoloides</i> Castellani.
<i>Escherichia paracoagulans.</i>	<i>Bacillus paracoagulans</i> Castellani, 1914.
<i>Escherichia paraentericus.</i>	<i>Bacillus paraentericus</i> Castellani, 1914.
<i>Escherichia pseudocoli.</i>	<i>Bacillus pseudocoli</i> Castellani, 1909.
<i>Escherichia pseudocoliformis.</i>	<i>Bacillus pseudocoliformis</i> Castellani, 1917.
<i>Escherichia pseudocolumben-</i> <i>sis.</i>	<i>Bacillus pseudocolumbensis</i> Castellani, 1917.
<i>Escherichia schaefferi.</i>	<i>Bacillus schaefferi</i> v. Freudenreich.
<i>Escherichia vekanda.</i>	<i>Bacillus vekanda</i> Castellani.
<i>Escherichia vesiculosus.</i>	<i>Bacillus vesiculosus</i> Henrici.
<i>Escherichia werahensis.</i>	<i>Bacillus werahensis</i> Castellani.
<i>Escherichia wesenbergi.</i>	<i>Bacillus wesenbergi</i> Castellani.
<i>Escherichia wesenbergoides.</i>	<i>Bacillus wesenbergoides</i> Castellani, 1916.
<i>Lactobacillus acidophilus.</i>	<i>Bacillus acidophilus</i> Moro, 1900.
<i>Lactobacillus acidophil-</i> <i>aërogenes.</i>	<i>Bacillus acidophil-aërogenes</i> Torrey and Rahe, 1915.
<i>Proteus asiaticus.</i>	<i>Bacillus asiaticus</i> Castellani and Chalmers, 1905.
<i>Salmonella archibaldi.</i>	<i>Bacillus archibaldi</i> Castellani and Chalmers, 1918.
<i>Salmonella guimai.</i>	<i>Bacillus guimai</i> Castellani and Chalmers, 1910.
<i>Salmonella morgani.</i>	<i>Bacterium morgani</i> Castellani and Chalmers, 1918.
<i>Salmonella veboda.</i>	<i>Bacillus veboda</i> Castellani, 1909.
<i>Salmonella watareka.</i>	<i>Bacillus watareka</i> Castellani.
<i>Salmonella woliniæ.</i>	<i>Bacillus woliniæ</i> Castellani, 1916.

Intestinal Non-Pathogenic Organisms.

Terms Used.

Aërobacter aërogenes.
Aërobacter cloacæ.
Bacillus subtilis.

Bacteroides bifidus.
Bacteroides fragilis.
Bacteroides oviformis.
Bacteroides ramosus.
Clostridium butyricum.

Lactobacillus acidophilus.
Proteus vulgaris.

Terms Discarded.

Bacterium lactis aërogenes Escherich, 1900.
Bacillus cloacæ, Jordan.
Bacillus subtilis Cohn.
Vibrio subtilis Ehrenberg.
Bacillus bifidus communis Tissier, 1900.
Bacillus fragilis Veillon and Zuber, 1898.
Coccobacillus oviformis Tissier.
Bacillus ramosus Veillon and Zuber.
Vibrion butyrique Pasteur, 1861.
Bacillus amylobacter v. Tieghem, 1877.
Bacterium navicula Reinke u. Berthold, 1878.
Bacillus butylicus Fitz, 1884.
Butylbacillus e Buchner, 1885.
Bacillus amylobacter I, Gruber, and *Bacillus amylobacter* II, Gruber, 1887.
Bacillus amylocyme Perdrix, 1891.
Bacillus butyricus Botkin, 1892.
Granulobacter saccharobutyricus Beijerinck.
Granulobacter saccharobutyricus Beijerinck, 1893.
Bacillus orthobutylicus Grimbert, 1893.
Amylobacter butylicus Duclaux, 1895.
Granulobacter butylicus Beijerinck, 1896.
Bacillus saccharobutyricus v. Klecki, 1896.
Motile Buttersaurebacillus Grassberger u. Schattenfroh, 1902.
Clostridium der Hanfrostes Behrens, 1902.
Clostridium pastorianum Winogradsky, 1902.
Plectridium plectinovorum Stormer, 1904.
Clostridium giganteum Kentner, 1904.
Clostridium americanum Pringsheim, 1906.
Granulobacter pectinovorum Beijerinck u. van Delden, and *Granulobacter urocephalum* Beijerinck u. van Delden, 1906.
Alcohol bildendes Clostridium of Schardinger, 1907.
Bacillus amylobacter Bredemann, 1909.

THE INTESTINAL PATHOGENIC ORGANISMS.

Alcaligenes Fæcalis.

This is a common organism in man and is presented as a slender bacillus about $\frac{1}{3} \mu$ in breadth and $2\frac{1}{2}$ to $3\frac{1}{2} \mu$ in length. They are frequently longer, measuring 7 to 10 μ in length.

They have a superficial resemblance to the *Eberthella typhi*, but are generally thinner and longer. They grow both aërobically and anaërobically without much difficulty on ordinary media. They present somewhat the pathogenicity of the *Eberthella typhi* on injection in small animals and are an important organism of identification and therapy in the work.

Incidence: Commonly found as straight and associated infections.

Therapy: Rectal and subcutaneous vaccines, ectoantigens, diet and irrigations.

Bacillus Cavicidus.

See table for cultural characteristics.

Incidence: Practically nothing is known of this bacillus.

Therapy: Suggested in above.

Bacillus Furcosus.

This organism is but rarely found in the intestinal canal, although it may occur, in which instance it would have significance. It is cultivated with difficulty, usually best in surface agar colonies. When found in feces its presence should be made note of.

Incidence: Infrequent. May be of biologic importance in connection with association.

Therapy. Subcutaneous vaccines; biologic considerations of associations.

Bacillus Fusiformis.

This organism occurs as thick, stubbed homogeneous organisms with round or pointed ends, usually appearing as single cells or in twos. They measure generally from 0.5 to 0.75 by 1.5 to 2 μ . They are active spore producers and belong to the sporulating organisms in the intestines of significance in this connection.

Incidence: A common infecting organism of the intestinal canal.

Therapy: Chlorine and other types of antiseptic enemas, and biologic considerations according to the associations; sera.

Clostridium Edematis Maligni.

This is the well-known organism *Vibrio septique* of Pasteur. This is a strict anaërobe developing in a vacuum or in the bottom of deep agar tubes. In my opinion, even though it be proteolytic, it is distinctly pathogenic in the intestinal canal of man where it not uncommonly is found as an infection. It occurs in rather small rods with rounded ends measuring $1\ \mu$ in thickness by $3\ \mu$ in length. There are usually variations in size as well as in shape. The organisms usually retain the Gram stain provided they are not treated with alcohol too long. See cultural characteristics.

Incidence: An important toxic organism commonly found in the intestinal canal.

Therapy: Rectal antagonisms according to the associations of the organism, and irrigations with chlorine, permanganate of soda, etc., solutions. Anaërobic sera.

Clostridium Putrificum.

This is presented as straight, thin rods in twenty-four hour broth cultures, measuring 0.6 to $0.8\ \mu$ in width by 5 to $6\ \mu$ in length. It is often curved, is an active spore producer, a strict anaërobe, and is one of the most important organisms in connection with the subject of intestinal toxemia. It has no saccharolytic properties, these being only proteolytic, and, therefore, is not mentioned in any significant sense under sugar reactions. It is easily cultivated in glucose agar, deep glucose agar, deep plain agar, surface agar, in gelatine, Loeffler's serum, plain broth, glucose broth, brain broth, milk, egg-meat and egg-white. Being a strong proteolytic organism it rapidly transforms native protein, usually, however, with the production of hydrogen sulphide, but no phenol or indol. It has been believed to be a non-pathogenic organism, but in connection with chronic intestinal toxemia it is one of the most significant organisms of the entire grouping, and when found in any numbers has great significance in a diagnostic and therapeutic sense. A number of varieties of the *Clostridium putrificum* have been described, at least six different strains.

Incidence: Commonly found as a straight or an associated infection.

Therapy: Non-protein diet, chlorine enemas, and biologic considerations according to the associations. Anaërobic sera.

Clostridium Tetani.

These in gelatine cultures a few days old are about 2 or 4 μ long and 0.3 to 0.5 μ wide, with slightly rounded ends. It is an active spore producer and is one of the most significant organisms in the intestinal canal in this connection. It is my belief that this has saccharolytic properties and feeble proteolytic ones. It is an organism that enjoys the widest sort of distribution in water and soil, and is an active toxin producer in the intestinal canal. The reader is referred to works on bacteriology for a full description, and to the table of cultural characteristics.

Incidence: As mentioned above.

Therapy: Chlorine and other types of antiseptic enemas, diet and biologic considerations according to the associations. Practically nothing is accomplished in the way of specific immunity production. Sera. Antagonisms.

Eberthella Belfastiensis.

These occur in rods, 0.5 by 1.0 to 1.5 μ , occurring singly and in pairs. They have significance in chronic infections, although it is not possible to be very definite about this. Occasionally they produce fever somewhat resembling typhoid, and in my experience have caused short diarrheic conditions. See table of cultural characteristics.

Incidence: Uncommonly found.

Therapy: Subcutaneous and rectal vaccines, and diet.

Eberthella Dispar.

This is a lactose-fermenting organism of the dysentery group originally described by Duval. The non-motile rods have rounded ends, occur singly and measure 0.5 by 1.0 to 1.5 μ in length. It clots and acidifies litmus milk usually in about

two weeks. It is acid in glucose, maltose, lactose, mannite, sometimes in saccharose and sometimes in dulcite. Its indol reaction is positive. Its relation to dysentery has not been clearly proven, although it is one capable of producing diarrheic states in man.

Incidence: Uncommonly found in chronic infections.

Therapy: Subcutaneous vaccines, ectoantigens, diet and irrigations.

Eberthella Douglassi.

For identification see cultural table.

Incidence: Common, either as a straight infection or as an associated infection.

Therapy: Subcutaneous vaccines; rectal vaccines; breaking up of association; high protein diet.

Eberthella Dysenteriae Shiga-Kruse.

This is the important organism throughout the world in cases of dysentery and in some of the more severe cases of diarrhea in this country. As years go on my cases of this type of dysenteric infection are being found from everywhere and it has significance in connection with this subject in that there are individuals who are chronic carriers, usually more or less diarrheic but sometimes not, in whom the organism has importance in connection with the subject. The organism is a plump, short bacillus with rounded ends, usually somewhat shorter and thicker than the *Escherichia typhi*. On plain agar in twenty-four hours it measures about $0.4\ \mu$ in breadth by 0.5 to $1.5\ \mu$ in length. For matters pertaining to its staining reactions, cultivation, cultural characteristics, etc., consult works on bacteriology.

As is well known it has a distinct virulent effect on man; the organisms in infected individuals commonly get in the blood stream and organs, especially the spleen. The organism produces an endocellular and an extracellular toxin. The endotoxin produces severe nerve lesions with paralysis, and the exotoxin produces the diarrhea, loss of weight and chronic intestinal conditions. This organism is commonly found in tox-

emia cases, oftentimes when one least expects to find it, and it has significance in connection with the diagnosis and therapy.

Incidence: While commonly looked for and seldom found it may be present as a chronic infecting organism in a non-diarrheic case of intestinal toxemia.

Therapy: When found, best cleared with antidysentery sera. Subcutaneous vaccines may be employed. Antagonism, several forms of *Escherichia coli*.

Eberthella Dysenteriae Y of Hiss and Russell.

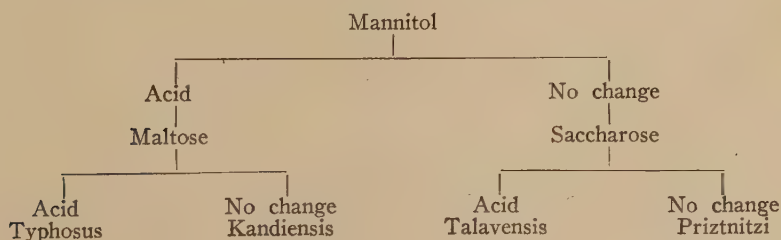
This organism resembles the Shiga bacillus morphologically and culturally. Its pathogenicity is like that of the Shiga but not so virulent. Its connection in this subject with a possible diarrhea case may have significance, but in the chronic affection it probably has little or no significance.

Incidence: As mentioned.

Therapy: Nothing much is known.

Eberthella Kandiensis.

This organism belongs to the genus *Eberthus* and to the type species of the *Eberthus typhosus*. The rods occur singly measuring 0.4 to 0.5 by 1.0 to 1.5 μ . They are motile, possessing peritrichous flagella. This genus has as its type species the organism which causes that variety of enteric fever which is called typhoid fever, as well as a number of species which are the causal agents of forms of diarrhea. The genus contains the following species in addition to the type, which may be differentiated biochemically as follows, though they can be distinguished in addition by their serological reactions:

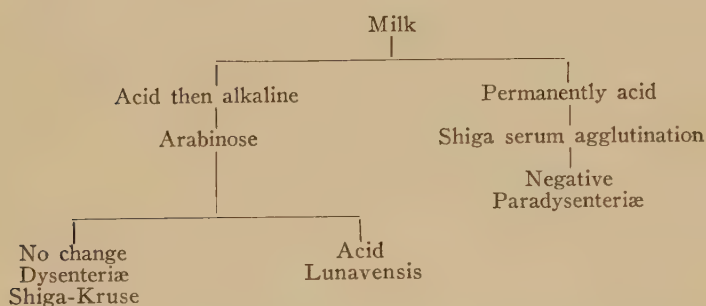


Incidence: This organism may be found as one of a chronic infection of the intestinal canal and has significance in identification and therapy.

Therapy: Subcutaneous vaccines, ectoantigens and the breaking up of bacterial associations. Lactose feedings.

Eberthella Lunavensis.

This organism belongs to the Shiga-Kruse division, mannitol not fermented organism, and may be best presented as follows:

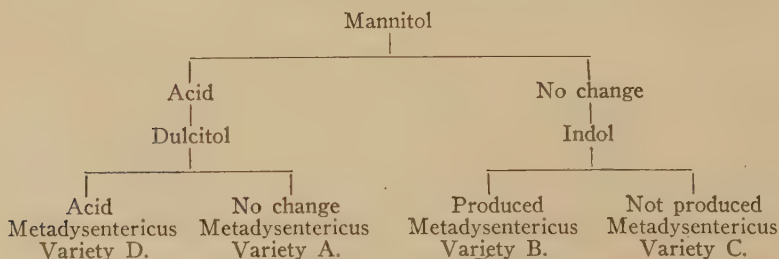


Incidence: Uncommonly found in chronic infections.

Therapy: Rectal vaccines, subcutaneous vaccines and the breaking up of bacterial associations.

Eberthella Metadysenteria, Varieties A, B, C and D.

This organism belongs to the genus of *Dysenteroides metadysentericus*, of which there are four forms differentiable as follows:



Incidence: All of these organisms are of distinct significance in connection with intestinal toxemia diagnosis and therapy, not only in connection with the specific infection that they produce, but more particularly in connection with the symbioses that are present with them. Occasionally the agglutination reaction may help in the proof of their presence and identification, but as a rule this has not been successful. It is required that they be identified directly from stool examination. This organism is a common producer of various low grades of colon pathology, practically all of which are today considered by the profession as only symptomatic conditions of the colon, and treated in all sorts of more or less useless and indirect ways.

Therapy: Subcutaneous vaccines, ectoantigens, sera, the breaking up of inimical bacterial associations by biologic methods as suggested by the infections, and careful dieting beginning with long periods of milk.

Eberthella Minutissima.

These occur in rods 0.5 by 1.0 μ , usually singly or in pairs. See table of cultural characteristics.

Incidence: But little information on hand.

Therapy: Negative.

Eberthella Paradysenteriae.

This organism is the well-known *Bacillus dysentericus* originally isolated by Flexner from cases of dysentery in the Philippine Islands. It differs somewhat in cultural features from the Shiga bacillus and can be distinguished from it by serological reactions. Morphologically this bacillus is about the same width as the Shiga bacillus, but the majority of the organisms are somewhat longer, measuring frequently 2 to 4 μ in length, and larger forms are common. The cultures on agar are like those of Shiga and on glucose litmus agar plates a permanent acidity is produced. Gelatine colonies are apt to be button-shaped, round and raised. The organism grows well in human bile and reduces nitrates to nitrites, some strains producing indol. Its pathogenicity is like that of the Shiga

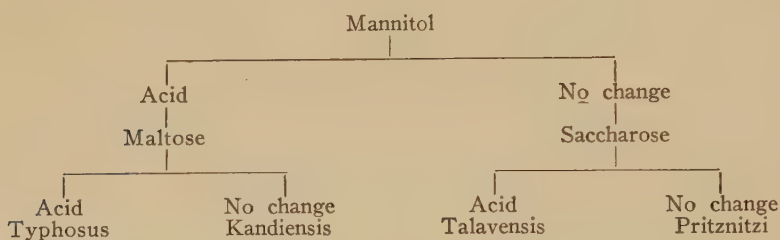
bacillus except that the virulence for laboratory animals is not so high, larger doses being required to kill. This organism may be met with at any time in intestinal toxemia work, even in the non-diarrheic cases, although not uncommonly a diarrhea history can be obtained.

Incidence: Occasionally found as chronic infections even in non-diarrheic conditions, although usually diarrhea is present.

Therapy: Rectal vaccines, subcutaneous vaccines, ectoantigens, sera and diet.

Eberthella Pritznitzii.

This organism belongs to the genus Eberthus, the type species being significant in connection with typhoid fever. It is best described as follows:

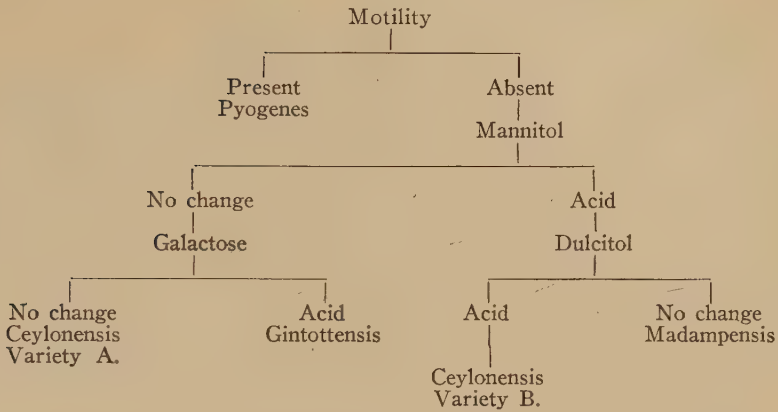


Incidence: It may be the cause of diarrheic states and congested and catarrhal conditions in the colon, and it is important in the work in identification and therapy.

Therapy: Rectal and subcutaneous vaccines, ectoantigens, the breaking up of biologic associations and diet.

Eberthella Pyogenes.

This represents the type organism of the genus Lankoides type, the species being best recognized and classified as below. This motile peritrichous flagellated organism occurs singly and is commonly found to measure 0.5 by 1.0 to 1.5 μ .

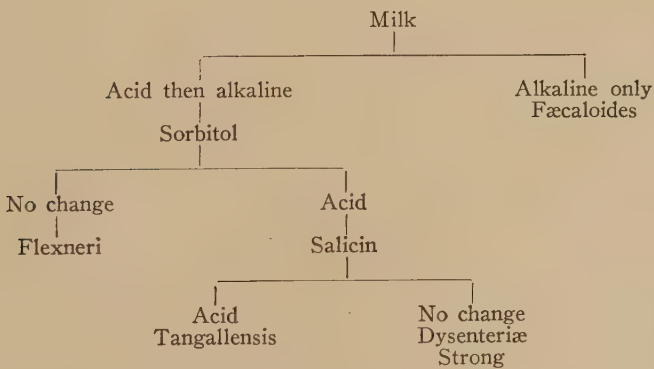


Incidence: Commonly found as a straight or associated infection.

Therapy: Subcutaneous vaccine, ectoantigens, sera.

Eberthella Tangallensis.

This organism is best described under the following schema:



Incidence: Commonly found as a cause associated with intestinal toxemia, and a producer of colonic pathology.

Therapy: Rectal and subcutaneous vaccines; ectoantigens; antagonisms, usually those of the *Escherichia coli* grouping, and diet.

Eberthella Typhi (Eberth).

This is the well-known etiological agent in typhoid fever. It is an organism somewhat more delicate than the *Escherichia coli*. On plain agar twenty-four hours old the organisms usually measure 0.4 to 0.5 μ in breadth and 1 to 3 μ in length, but longer forms measuring 4 to 5 μ in length are not uncommon. In some strains the organism is differentiated morphologically from the *Escherichia coli* with great difficulty. The staining reactions, cultivation, cultural characteristics, special media, etc., as they pertain to the condition known as typhoid fever, will not be presented in this book. While chronic carriers are considered of interest in medicine only because of their liability of infecting others with the acute infection (typhoid fever), typhoid carriers are more common than the profession believes, and the constant presence of the organism in the intestinal canal is an important one in connection with the subject of this volume.

All chronic carriers are distinctly toxic, the effect clinically being more particularly produced by the hemotoxin type. As a rule agglutinins are present in the blood of infected individuals, this being found also in the chronic carrier, which probably represents about 4 per cent. of all those who have been infected at one time with typhoid fever. Of course most of these carriers are short-termed, because of the stool becoming negative 3 or 6 months after the initial attack, but this does not represent all instances by any means, because chronic infections of this organism, which seemingly is capable of a toxemia even independent of various symbiotic associations, is an important item in the diagnosis and therapy of this subject.

Incidence: In chronic infections following typhoid fever not infrequently found. It is supposed to be innocent in this connection, but is a common producer of pathology in the intestine and chronic states of intestinal toxemia.

Therapy: Subcutaneous vaccines, ectoantigens, diet and sera.

Erythrobacillus Prodigiosus.

This is a small bacillus appearing as a micrococcus, usually about $0.5\ \mu$ broad and 0.5 to $1\ \mu$ long. It may occur in short chains, and regarding this organism, for which there are many synonymous terms, much is known in its connection with chronic intestinal toxemias, in which it is a distinct offender, although it is believed by many to be essentially an innocent organism in the intestinal canal. Its significance, however, is more particularly in association with other organisms, many of which it symbioses with readily. There are a number of varieties, some fermenting saccharose as well as dextrose, other forms fermenting dextrose, saccharose and lactose. One of the peculiar features of most all of the strains is its pigment-producing ability. See cultural characteristics.

Incidence: Commonly found in straight and associated infections of slight degree.

Therapy: Subcutaneous vaccines, ectoantigens, biologic considerations according to the associations, antagonisms.

Escherichia Carolinus.

This organism belongs to the type genus *Balkanella* described by Castellani and Chalmers in 1918, in which there are two types, the *carolinus* separated from the other by the production of acid and gas on saccharose, thus distinguishing it from the *Balkanella coagulans*, the latter of which represents the type of species. It has been discovered in the blood in cases of diarrhea. It can be separated by specific serum reaction from the *Eberthus typhosus*, *Salmonella paratyphi*, *Salmonella paratyphosa* and *Salmonella gaertneri*.

Incidence: Occasionally found in acute, subacute and even diarrheic conditions.

Therapy: Subcutaneous vaccines, ectoantigens, rectal instillations of antagonisms, *Escherichia coli* 10 to 17, colonic washings—warm antiseptic alkaline solutions.

Escherichia Coagulans.

This organism belongs to the genus *Balkanella*, it being distinguished from the *carolinoides* by the production of no change on saccharose. It belongs to the type of species of *Wesenbergus wesenbergi*. There is still a further identification of the genus *Balkanella* in the *Wesenbergus fermentosus*, in which the species may be separated in those that produce indol, namely (1) dulcitol fermented, *Wesenbergi*, (2) dulcitol not fermented, *Giumai*; and in the *B.* strains that do not produce indol, namely *Fermentosus*.

Incidence: Not uncommonly found and of importance in associated infections.

Therapy: Subcutaneous vaccines, ectoantigens, biologic restrictive actions by other organisms of the *Escherichia coli* group, mainly in the *Communion* class.

Escherichia Coli.

This is the most common organism of the intestinal canal found normally in man and practically all animals as well as in various locations in the environment, as in soil, water, sewage, food, etc. It has been described under a great variety of names, especially as the cause of a number of diseases in animals, in which commonly it is the secondary invader and not the etiological agent of the disease in question. It possesses a four-fold significance in the intestinal canal of man, first, it is an inhibitor in some species and not a few of the inimical organisms; second, it may act as an infecting agent in some species; third, its presence may be of negative significance; and fourth, it can act as a toxin producer in association with other organisms, mainly the anaërobes, in which the anaërobes prepare the pabulum for these organisms to split down into toxic bodies. It is a small bacillus, measuring on plain agar in twenty-four hours 0.4 to 0.6 μ in width by 1 to 2 μ in length. It usually is a short oval or almost rounded organism, although it may appear almost like diplococci. Different strains differ in morphology. Certain strains are encapsulated and free from motility. For the many points in connection with this

Genus Escherichia Coli Group.

Dextrose +
Lactose +

Dulcitate +
B. communior
B. communis

Dulcitate —
B. aërogenes
B. acidilactici

Saccharose +
B. communior

Saccharose —
B. Communis

Saccharose +
B. aërogenes

Saccharose —
B. acidilactici

Type 1 Mannite + Raffinose +
Type 2 Mannite + Raffinose —
Type 3 Mannite + Raffinose —
Type 4 Mannite — Raffinose +
Type 5 Mannite — Raffinose —

Type 6 Mannite + Raffinose +
Type 7 Mannite + Raffinose —
Type 8 Mannite + Raffinose +
Type 9 Mannite — Raffinose —

Type 10 Mannite + Raffinose +
Type 11 Mannite + Raffinose —
Type 12 Mannite — Raffinose +
Type 13 Mannite — Raffinose —

Type 14 Mannite + Raffinose +
Type 15 Mannite + Raffinose —
Type 16 Mannite — Raffinose +
Type 17 Mannite — Raffinose —

organism the reader is referred to any of the text-books on bacteriology.

The pathogenicity depends somewhat upon the original strain and also upon its association with other organisms. As a rule freshly isolated strains are virulent to guinea-pigs and rabbits and somewhat less so to mice. Any of the strains which occur in diarrheic conditions are usually of the parasitic type, especially where definite reasons are present, whereas the strains from normal stools as a rule are distinctly less virulent and may be negative or of the inhibiting type. The organism loses its virulence quickly on artificial cultivation. When associated in the intestinal canal with streptococci and staphylococci, or with any of the anaërobes, it has distinct significance in connection with intestinal toxemia. Any of the virulent strains of this organism escaping from the intestinal canal may cause pathology with septic conditions.

One occasionally can accomplish significance of the type present in a case by the agglutination reaction. It must be remembered, however, that not uncommonly in distinct infections of this organism examination of the blood may be quite worthless. A most important feature in connection with this organism is the identification of various strains on their different reactions to carbohydrates. The following is a schema of value in this connection, the parasitic forms of which are most often found in numbers 6, 7, 8 and 9.

Incidence: Common. Important in straight infections in those of the parasitic type, and in associated infections. Other types, especially those of the *Communior* variety, are distinctly inhibitive to many forms.

Therapy: Vaccines, rectal and subcutaneous, ectoantigens, and antagonisms, *B. prodigiosus* and *pseudomonas*, sometimes *B. welchii*, practically all organisms of the lactic acid producing type. Non-protein types of diet.

Escherichia Communior.

This organism, measuring 0.5 to 0.6 by 1.0 to 3.0 μ in length, is identifiable from the well-known *Escherichia coli*

because of its positive action on saccharose, which it ferments. It is a far more common variety of this group than the *Escherichia coli* and is one of the innocent and restrictive types of organisms of the intestinal canal. However, there are undoubtedly certain strains which are inimical to the host, these almost always being bound up in various symbiotic connections with other organisms. There are instances in which the organism possesses a distinct pathogenicity to the human being. It has importance in diagnosis and in therapy.

Incidence: A common denizen of the human intestinal canal, in which these organisms act vigorously in an antagonistic way through a group of organisms.

Therapy: When parasitic, subcutaneous and rectal vaccines, and ectoantigens; when innocent, best left alone when found and usually has to be added to.

Escherichia Grünthali.

This organism occurs as rods 0.5 to 0.7 by 1.0 to 1.5 μ , always singly.

Incidence: Found apparently only in cases of poisoned "liver paste."

Therapy: Not important in this connection.

Escherichia Leporis.

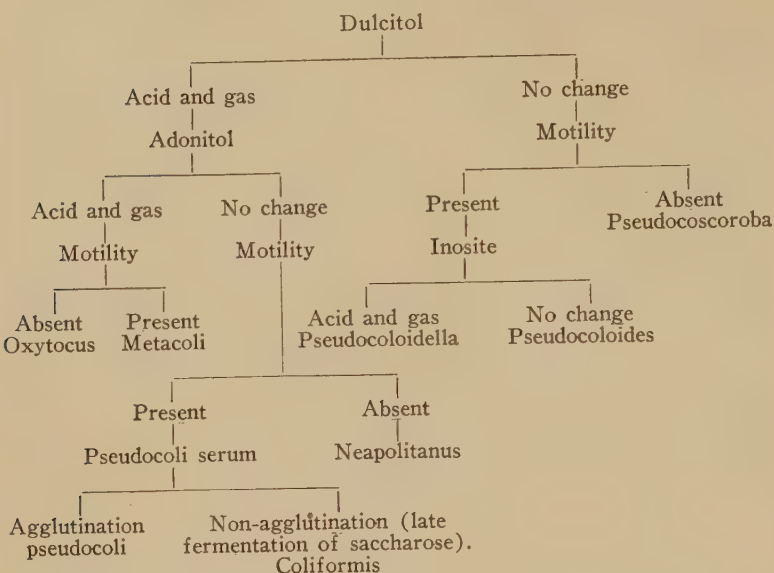
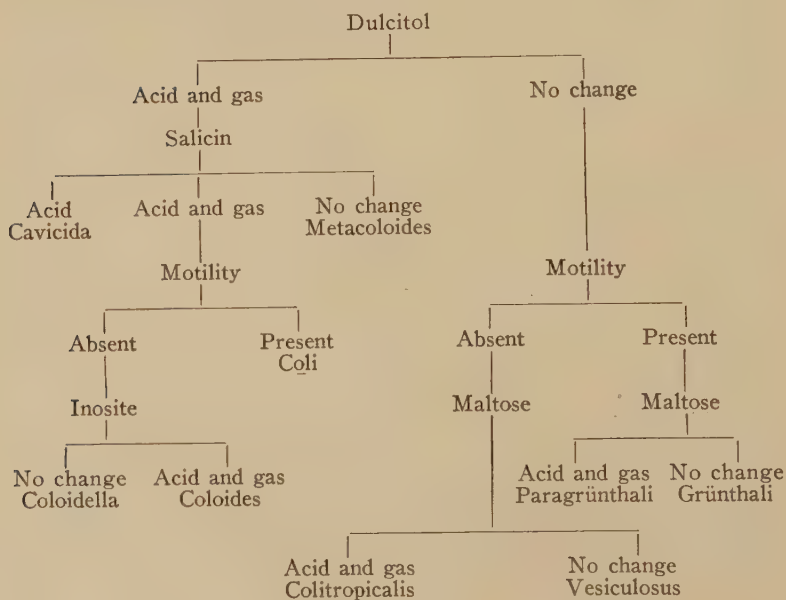
This occurs in rods, 0.5 by 4.0 to 6.0 μ , usually singly, and is an organism of significance. See table of cultural characteristics.

Incidence: Uncommonly found.

Therapy: Very little is known other than attacking along the lines of bacterial associations.

Escherichia Paragrünthali.

This organism, occurring singly, measuring 0.5 to 0.6 by 1.0 to 3.0 μ , being motile by peritrichous flagella, is best described in the following schema of Smith's Indol-Producing Division:

Durham's Saccharolytic Communior Section.*Durham's Non-saccharolytic Communis Section.*

Incidence: It may therefore be seen that it belongs to the non-saccharolytic communis section and in my experience is an organism of significance in connection with work mainly as a secondary toxin producer, and requiring attention like most of the organisms of the communis section in both identification and attention, specifically in therapy. Important in associated infections, persisting in inimical symbiotic groupings.

Therapy: Rectal and subcutaneous vaccines, ectoantigens, the breaking up of inimical biologic associations, diet and irrigations.

Pneumococcus.

This well-known organism in the production of pneumonia is often found in the intestinal canal, from which it is capable of passing into the blood stream and causing various lesions in the lungs, middle ear, mastoids, meninges, endocardium, pericardium, joints, kidneys and peritoneum. Description of this well-known organism need not be offered.

Incidence: Commonly found in associated infections.

Therapy: Subcutaneous vaccines, ectoantigens, antipneumococci sera, antagonisms, and attention to bacterial associations. Transduodenal lavages or enemas of permanganate of potassium 1:6,000-12,000. Antagonisms.

Proteus Valeriæ.

These occur in rods 0.5 by 1.0 to 1.5 μ , usually singly or in pairs and may represent the chronic infection of the human intestinal canal of significance. See table of cultural characteristics.

Incidence: Occasionally found as a straight or mixed infection.

Therapy: Biologic considerations of the associated infection, and irrigations.

Proteus Vulgaris.

This is a common denizen of the intestinal canal of man and occurs as a thin bacillus measuring about 0.5 to 0.6 μ by 1 to 1½ μ on plain agar twenty-four hours old. Many long

forms are also present, measuring 2 to 3 μ in length, and threads are common, some of them measuring as much as 20 μ . This organism has an energetic proteolytic action in the intestinal canal. It is not a putrefactive organism like the anaërobes of the *B. sporogenes* and *B. histolyticus*, but when it occurs in proper symbiosis it possesses considerable significance in connection with this subject. When actively growing in the intestinal canal it is capable of producing distinct degenerative states in the body. It is capable of wide invasion from the intestinal canal and is important as a secondary invader. It can, however, produce a chronic form of cystitis without associations. Its identification and attention are important in this work.

Incidence: This organism is supposed to be innocent in the intestinal canal, but it is definitely toxic and more particularly in association with other infecting organisms. It has a wide distribution.

Therapy: Rectal and subcutaneous vaccines, biologic considerations according to the associations, diet and irrigations. Antagonisms.

Pseudomonas Aëruginosa.

This organism was first described as identical with the *Bacillus pyocyaneus* and is important in connection with intestinal toxemia. It occurs as thin rods, sometimes curved, measuring 0.25 by 3 to 4 μ , from twenty-four hour agar cultures when stained by gentian-violet. In many instances they are longer and often grow into long threads. Much is known regarding their cultural characteristics. The reader is referred to works on bacteriology for full descriptions. It produces proteolytic ferments, although the breaking down of protein does not go as far as peptone production.

It is an active hemotoxin producer, and is distinctly of pathogenic significance in the intestinal canal of man. It possesses significance in the intestinal canal in that when it occurs in any quantity staphylococci and streptococci are practically never found. In this sense it is distinctly antagonistic to these organisms as well as to the *Bacillus anthracis*, which is not an

uncommon denizen in the intestinal canal of man. In this sense the *Bacillus pyocyaneus* is a valuable organism in an antagonistic way against the above-named infection. In itself, however, it is distinctly an etiological agent producing distinct inflammatory conditions in the bowel as well as mild states of pathology. While this organism in the intestinal canal of man has been judged as of little significance, in my opinion it represents one of the distinct organisms of chronic infectivity. See cultural characteristics.

Incidence: As mentioned above.

Therapy: Subcutaneous vaccines, ectoantigens, biologic considerations according to the associations. Antagonisms—strains of streptococci and staphylococci.

Pseudomonas Non-liquefaciens.

These occur as short slender rods with rounded ends, usually singly. Not much is known about this organism in its significance in the intestinal canal. I believe that in most instances it is one of little or no pathologic significance. However, there have been infections met with in which this organism was prominent and therefore it should be mentioned in this connection.

Incidence: Negative.

Therapy: Negative.

Salmonella Aërtrycke.

This organism is found in epidemics of meat poisoning and usually is derived from veal of calves suffering from a chronic enteritis. Since it is capable of intense immunity reaction it has not been found in examinations of stools by me, probably because of this intense immunity reaction in the blood. The bacteria have round ends, occur often in pairs, 0.6 to 0.8 μ in length, and grow with abundance of gas on glucose agar, the growth not being coagulated for several weeks. They form a turbidity rapidly in alkaline bouillon with the surface pellicle easily dislodged. The growth as a rule is odorless and no indol is produced. It is virulent to mice, guinea-pigs

and rabbits in doses of 0.1 c.c., killing them generally in from 3 to 6 days. As a rule it rapidly produces agglutinin in the blood, in certain individuals this agglutination running as high as 1 to 1000.

Incidence: Found in epidemics of food poisoning.

Therapy: Nothing is known in a biologic way.

Salmonella Enteritidis.

This organism was discovered by Gaertner in cases of meat poisoning from the use of infected meat from cattle. It is a small bacillus measuring 0.3 to 0.4 μ in diameter, and 1 to 2 μ in length on plain agar twenty-four hours old, together with many short, almost coccid organisms. Large forms are not uncommon, measuring as much as 0.6 to 0.7 by 3 to 4 μ . There are no spores. The motility is usually marked, but non-motile forms have been isolated. On plain agar the surface colonies are small and delicate, older ones being much larger. It has certain cultural characteristics, for which see works on bacteriology.

The organism is pathogenic to small animals, different strains differing considerably in virulence. It produces a soluble poison which loses its activity rapidly. For this reason agglutinins are usually found in the blood, although because of the difference in the strains and groupings and association, such testing for the infection may prove erroneous, especially in that the organism is agglutinated by other intestinal organisms, the most notable being the typhoid bacillus. The organism possesses considerable significance in connection with intestinal toxemia, more particularly in the more acute conditions. However, it can be found and often is found in symbiotic association with the organisms of the typhoid dysenteric groupings and possesses specific significance in the biologic handling of cases in which it is present.

Incidence: Practically never found in chronic intestinal infections, usually only in meat poisoning from the use of infected meats from cattle.

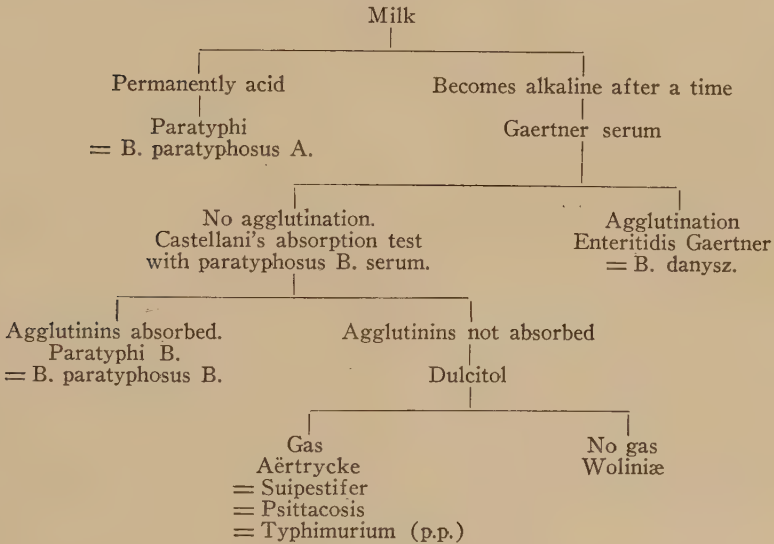
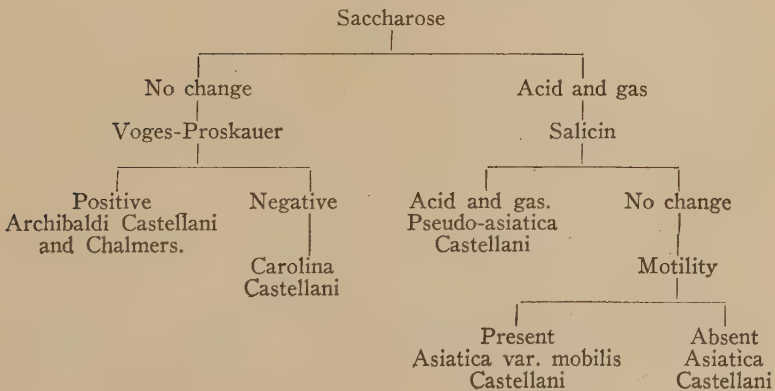
Therapy: That of the acute toxic condition.

Salmonella Mobilis.

This organism is best described in the schema presented in the Paratyphoid-asiaticus group of the genus Salmonella.

A. Indol not produced—Paratyphoid division.

B. Indol produced—Asiaticus division.

Paratyphoid Division—Indol Non-producers.*Asiaticus Division—Indol Formers.*

Incidence: Occasionally found as the cause of straight and associated infections, even in non-diarrheic conditions. A producer of chronic colon pathology.

Therapy: Rectal vaccines, subcutaneous vaccines, the breaking up of biologic bacterial associations and dieting.

Salmonella Negombensis.

For identification see cultural characteristics table.

Incidence: Common producer of intestinal toxemia as a straight or associated infection, and a producer of acute, sub-acute and chronic pathology.

Therapy: Rectal vaccines, ectoantigens, breaking up of bacterial associations, the use of antagonisms mostly derived from the *Escherichia coli* grouping, diet.

Salmonella Para-aërtrycke.

For cultural characteristics, see identification table.

Incidence: Common producer of intestinal toxemia, in straight or associated infections, and a producer of acute, sub-acute and chronic pathology.

Therapy: Rectal vaccines, breaking up of bacterial associations, the use of antagonisms mostly derived from the *Escherichia coli* grouping, diet.

Salmonella Para-asiatica.

See table of cultural characteristics.

Incidence: Occasional producer of intestinal toxemia, in straight and associated infections, and a producer of acute, sub-acute and chronic pathology.

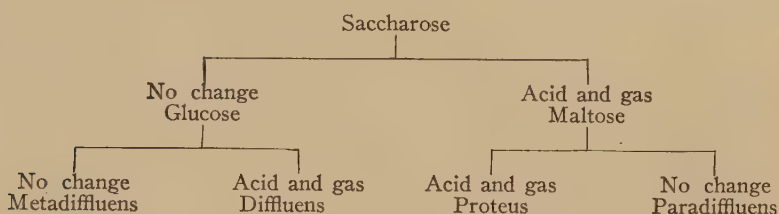
Therapy: Rectal vaccines, breaking up of bacterial associations; the use of antagonisms mostly derived from the *Escherichia coli* grouping, diet.

Salmonella Paradiffluens.

This organism belongs to the proteus tribe, the type species of which is the *Proteus vulgaris*. The tribe may be divided into genera as follows:

- A. Rapid gelatine liquefiers; do not ferment lactose; mostly Gram-positive—*Proteus*.
- B. Slow gelatine liquefiers; ferment lactose; Gram-negative—*Cloaca*.

This organism, as well as all of the *Proteus-Cloaca* group is difficult of classification, and serological reactions are of no value in their identification. Some of the species as presented by Castellani are as follows:



Incidence: While these organisms are considered more or less innocent forms, they possess much significance in symbiotic association in the intestinal canal. In my opinion they are far from as innocent as they are supposed to be. Their identification and handling in connection with this subject has considerable importance. Common producer of intestinal toxemia, in straight and associated infections, and a producer of acute, subacute and chronic pathology.

Therapy: Subcutaneous vaccines, breaking up of symbiotic groupings by antagonisms rectally or subcutaneously.

Salmonella Pseudo-asiatica.

The type species of this organism is the *Salmonella paratyphi* and is best described in the schema presented in connection with the genus *Salmonella*. In connection with this see schema under *Salmonella mobilis*.

Incidence: Occasionally found as the cause of straight and associated infections, even in non-diarrheic conditions. A producer of chronic colon pathology and toxemias.

Therapy: Rectal vaccines, subcutaneous vaccines, the breaking up of biologic bacterial associations, and dieting.

Salmonella Pseudomorgani.

See table for cultural characteristics.

Incidence: Occasionally found as the cause of straight and associated infections, even in non-diarrheic conditions. A producer of chronic colon pathology.

Therapy: Rectal vaccines, subcutaneous vaccines, ecto-antigens, the breaking up of biologic bacterial associations, sera and dieting.

Salmonella Pseudowesenbergi.

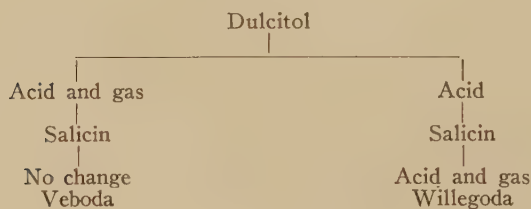
For characteristics of this organism, see cultural table.

Incidence: Occasionally found as the cause of straight and associated infections, often in non-diarrheic conditions. A producer of chronic colon pathology.

Therapy: Rectal vaccines, subcutaneous vaccines, ecto-antigens, the breaking up of biologic bacterial associations, and dieting.

Salmonella Willegoda.

This organism of the Veboda group is best described in the schema found in connection with the genus *Salmonella*.

Veboda Group.

Incidence: Commonly found in associated infections.

Therapy: Rectal and subcutaneous vaccines, ectoantigens, biologic considerations in breaking up symbiotic cliques.

Salmonella Schotmüller A.

This organism is known commonly as *Bacillus paratyphosus* A, which is differentiated from the paratyphoid B. The

organisms differ culturally and serologically from paratyphoid B, being much more rare. They are small, rather thin, bacilli, measuring 0.3 to 0.4 μ in width by $1\frac{1}{2}$ to 2 μ in length, and sometimes longer forms, measuring $2\frac{1}{2}$ to 3 μ . They are pathogenic to small animals, producing specific agglutinins in the blood stream. There is considerable confusion in regard to the differentiation of *Bacillus paratyphosus* A organism from paratyphoid B, and the failure to ferment xylose may be the only cultural distinction of value. It not uncommonly occurs as a chronic infecting organism in man, necessitating identification and specific treatment.

Incidence: A common organism producing toxemia and chronic colonic pathology.

Therapy: Rectal and subcutaneous vaccines, ectoantigens, the breaking up of bacterial associations, sera, diet and antagonisms.

Salmonella Schotmüller B.

This is a thin organism measuring 0.3 to 0.4 μ in breadth and 1 to 2 μ in length on plain agar twenty-four hours old. For cultural characteristics see table and works on bacteriology. It is distinctly more resistant than the typhoid bacillus, surviving for a long time, and is far more frequently present in chronic toxic states than its brothers A and C (the latter of which is no doubt a different organism from A and B, and possesses the same significance as they do in this work). This organism, like the other forms, is important of identification in the diagnosis of intestinal toxemia, and it not infrequently occurs as a chronic infection, produces distinct toxic states in the individual, and likewise such conditions as cystitis, pyelitis, bronchitis and pleurisy, arthritis, abscess in the joints, myositis, phlebitis, orchitis, lung abscess, otitis media, periostitis, osteomyelitis, gall-bladder infections, etc.

The organism is commonly taken in by food such as milk, cheese, salads, preserves and fish. As a rule the body undertakes to protect itself against the presence of the organism by the development of immune bodies, but while agglutination has been largely used in the classification of the strain the re-

sults are very difficult to interpret in this connection. The main differentiation is by means of reaction to separate the paratyphoids from the Gaertner group in bacteriolysis. A serum made from Gaertner dissolves Gaertner, but not paratyphoids. Their identification in this connection in the intestinal canal is important.

Incidence: Mentioned in Variety A.

Therapy: Mentioned in Variety A.

Staphylococcus Albus.

This organism, whose spheres measure 0.6 to 0.8 μ and occur singly, in pairs and in irregular groups, was originally described as the *Staphylococcus pyogenes albus*, its cultural reactions and pyogenic properties being practically identical with those of the *Staphylococcus aureus*. It is the common denizen or inhabitant of the skin of man as well as the mucous membrane, in which instances it is devoid of pathogenic action. In the intestinal canal, however, it has considerable significance in infectivity both in straight and mixed types of infection. See cultural characteristics.

Incidence: Commonly found in the intestinal canal. It may be a straight or an associated infection.

Therapy: Subcutaneous vaccines, ectoantigens and biologic considerations according to the associations. Antagonism—*Pseudomonas aëruginea*.

Vibrio Comma.

This is the etiologic factor of Asiatic cholera and need not be described in this connection. For further information regarding this organism, which is well known, the reader is referred to text-books on bacteriology. It does not enter into the production of intestinal toxemias of the type that are here described.

Incidence: Not found in chronic intestinal states.

Therapy: Therefore not important.

Vibrio Gindha.

This is a fairly long, definitely curved, rodlike bacillus, larger than the cholera vibrio, having single terminal flagella and cultural characteristics like those of the cholera vibrio. It is differentiated from the cholera vibrio by immunity reactions. Here and there in a diarrheic case these organisms may be found, but they possess no special significance in this connection.

Incidence: Very seldom found.

Therapy: Diet and irrigations.

Vibrio Maszauah.

This organism is one that is found in the dejecta of some patients with cholera-like symptoms, although it does not seem to be definitely associated with epidemics of cholera. It belongs to the etiology of cholera-like disorders and may even be more virulent than the cholera vibrio. It is not found in connection with chronic intestinal toxemias.

Incidence: Not found in connection with chronic intestinal toxemias.

Therapy: Not important.

The Streptococci.

Streptococci are the most important parasites of the human body. They occur in short or long chains, sometimes in pairs and small groups, never in large masses. They are variously classified, but in this connection the classification of Holman is used, this being the most elaborate and in many respects the most satisfactory classification so far advanced, and one which takes in a very large number of streptococci from a variety of sources. This classification uses hemolysis and fermentation of lactose, mannite and salicin as differential characteristics, since all streptococci ferment dextrose. He separates 16 main types as follows:

1. <i>Streptococcus infrequens</i> .	Hemolytic.	Ferments lactose, mannite, and salicin.
2. <i>Streptococcus hemolyticus</i> .	Hemolytic.	Ferments lactose and mannite, but not salicin.
3. <i>Streptococcus pyogenes</i> .	Hemolytic.	Ferments lactose and salicin, but not mannite.
4. <i>Streptococcus anginosus</i> .	Hemolytic.	Ferments lactose, but not salicin or mannite.
5. <i>Streptococcus hemolyticus</i> II.	Hemolytic.	Ferments mannite and salicin, but not lactose.
6. <i>Streptococcus hemolyticus</i> III.	Hemolytic.	Ferments mannite, but not salicin or lactose.
7. <i>Streptococcus equi</i> .	Hemolytic.	Ferments salicin, but not mannite or lactose.
8. <i>Streptococcus subacidus</i> .	Hemolytic.	Does not ferment lactose, mannite or salicin.
9. <i>Streptococcus faecalis</i> .	Non-hemolytic.	Ferments lactose, mannite, and salicin.
10. <i>Streptococcus non-hemolyticus</i> I.	Non-hemolytic.	Ferments lactose and mannite, but not salicin.
11. <i>Streptococcus mitis</i> .	Non-hemolytic.	Ferments lactose and salicin, but not mannite.
12. <i>Streptococcus salivarius</i> .	Non-hemolytic.	Ferments lactose, not mannite or salicin.
13. <i>Streptococcus non-hemolyticus</i> II.	Non-hemolytic.	Ferments mannite and salicin, but not lactose.
14. <i>Streptococcus non-hemolyticus</i> III.	Non-hemolytic.	Ferments mannite, but not salicin or lactose.
15. <i>Streptococcus equinus</i> .	Non-hemolytic.	Ferments salicin, but not mannite or lactose.
16. <i>Streptococcus ignavus</i> .	Non-hemolytic.	Does not ferment lactose, mannite or salicin.

Streptococcus Infrequens.

Often found in the human intestinal tract and of distinct pathogenic significance.

Incidence: Often found.

Therapy: Subcutaneous vaccines, ectoantigens, biologic considerations according to the associations, antagonisms.

Streptococcus Hemolyticus.

Commonly found in the human intestinal tract and of distinct pathogenic significance.

Incidence: Commonly found.

Therapy: Subcutaneous vaccines, ectoantigens, biologic considerations according to the associations, and antagonisms.

Streptococcus Pyogenes.

See Streptococcus infrequens and cultural characteristics. Often found in the human intestinal tract, is the best known of all streptococci, and distinctly of pathogenic significance.

Incidence: Often found.

Therapy: Subcutaneous vaccines, ectoantigens, biologic considerations according to the associations, and antagonisms.

Streptococcus Angiosus.

Occasionally found in the human intestinal tract and of pathogenic significance, especially in associations.

Incidence: Occasionally found.

Therapy: Subcutaneous vaccines, ectoantigens, biologic considerations according to the associations, and antagonisms.

Streptococcus Hemolyticus II.

Occasionally found in the human intestinal tract and in some instances of pathogenic significance.

Incidence: Sometimes found.

Therapy: Subcutaneous vaccines, ectoantigens, biologic considerations according to the associations, and antagonisms.

Streptococcus Hemolyticus III.

Occasionally found in the human intestinal tract and in some instances of pathogenic significance.

Incidence: Sometimes found.

Therapy: Subcutaneous vaccines, ectoantigens, biologic considerations according to the associations, and antagonisms.

Streptococcus Equi.

Occasionally found in the human intestinal tract and but rarely of pathogenic significance.

Incidence: Sometimes found.

Therapy: Subcutaneous vaccines, ectoantigens, biologic considerations according to associations, and antagonisms.

Streptococcus Subacidus.

Occasionally found in the human intestinal tract and of significance in associations.

Incidence: Sometimes found.

Therapy: Subcutaneous vaccines, ectoantigens, biologic considerations according to the associations, and antagonisms.

Streptococcus Fæcalis.

Generally found in human intestinal tract and may be of none, of association type, and occasionally of pathogenic significance.

Incidence: Generally found.

Therapy: Subcutaneous vaccines, ectoantigens, biologic considerations according to the associations, and antagonisms.

Streptococcus Mitis.

Often found in the human intestinal tract and in my opinion of pathologic significance.

Incidence: Often found.

Therapy: Subcutaneous vaccines, ectoantigens, biologic considerations according to the associations, and antagonisms.

Streptococcus Salivarius.

Often found in the human intestinal tract and in my opinion of pathologic significance in rare instances.

Incidence: Often found.

Therapy: Subcutaneous vaccines, ectoantigens, biologic considerations according to the associations, and antagonisms.

Streptococcus Non-hemolyticus I.

Often found in the human intestinal tract and of pathogenic significance.

Incidence: Often found.

Therapy: Subcutaneous vaccines, ectoantigens, biologic considerations according to the associations, and antagonisms.

Streptococcus Non-hemolyticus II.

Often found in the human intestinal tract and of pathogenic significance.

Incidence: Often found.

Therapy: Subcutaneous vaccines, ectoantigens, biologic considerations according to the associations, and antagonisms.

Streptococcus Non-hemolyticus III.

Often found in the human intestinal tract and of pathogenic significance.

Incidence: Often found.

Therapy: Subcutaneous vaccines, ectoantigens, biologic considerations according to the associations, and antagonisms.

Streptococcus Equinus.

Sometimes found in the human intestinal tract and in my opinion of pathologic significance in rare instances.

Incidence: Sometimes found.

Therapy: Subcutaneous vaccines, ectoantigens, biologic considerations according to the associations, and antagonisms.

Streptococcus Ignavus.

Sometimes found in the human intestinal tract and occasionally of pathogenic significance.

Incidence: Sometimes found.

Therapy: Subcutaneous vaccines, ectoantigens, biologic considerations according to the associations, and antagonisms.

Of the above streptococci pathogenic to man the best known is the *Streptococcus pyogenes*; the supposedly non-pathogenic

mouth streptococci belong to the *Streptococcus mitis* and *salivarius*; the streptococci found in scarlet fever to *Streptococcus anginosus*; the common intestinal streptococci to the *Streptococcus faecalis*, and the supposedly non-pathogenic streptococci from horse dung to the *Streptococcus equinus*. This classification links up the pathogenic properties and source with cultural reactions. It is the only one today based upon cultural characters which has practical value. Occasionally streptococci are found which do not fit the classification, but the types which Holman has worked out seem to be definite. Various strains of streptococci have various degrees of virulence, this also being present in different type strains.

It may here be said definitely that so far as any of the strains of streptococci are concerned they practically all are inimical in the intestinal canal of man. And it is from the intestinal canal that the vast majority of streptococcic infections occurring in various parts of the body are derived. Sir William Osler once said that to understand syphilis is to understand half of what there is to medicine. The same simile can be used in connection with streptococcus. To understand the streptococcus is to understand half of the acute, subacute and chronic infectious disorders of man, and more particularly a large number of pathological conditions of chronic nature which ensue from their toxic products. It represents the most important infecting organism in the intestinal canal and requires a very sharp interest at all times in the examination of stools.

The foregoing represent the pathogenic organisms found in the intestinal canal of man which are significant in connection with intestinal toxemia, the gut as a source of focal infection and especially as a source of toxin absorption. There remain a number of organisms which are variously classified, most often under pathogens, regarding which little is known as to whether they have special significance or represent synonymous terms with those already mentioned. Since in my belief and experience I have not been able to attach much if any significance to them, they are here merely mentioned:

Bacillus of Bovet.	Bacillus paratyphus Archard and Bensaud.
Bacillus coli concentricus Fitzpatrick.	Bacillus porceloides Roger and Garnier.
Bacillus coli icteroides Fitzpatrick.	Bacillus of Uptadel.
Bacillus coprogenes parvus Bienstock, 1886.	Bacteroides fragilis.
Bacillus dysenteriae Chantamesse and Vidal.	Bacteroides ramosus.
Bacillus dysenteriae liquefaciens Ogata, 1892.	Vibrio insolitus Castellani.
Bacillus monadiformis Messea.	Vibrio kegalliensis var. paracholerae Castellani.
Bacterium tholoideum Gessner.	

THE INTESTINAL SAPROPHYTIC ORGANISMS.

Achromobacter Metadiffuens.

This organism belongs to the genus *Proteus* and is best described in the schema under the *Salmonella paradiffuens* organism.

Incidence: It may occur as a straight or as an associated infection.

Therapy: Subcutaneous vaccines, breaking up of symbiotic groupings by antagonisms rectally and subcutaneously.

Achromobacter Paradyserteriae.

This organism belongs to the subgenus *Flexnerella* in the mannitol not fermented group and is best described in the schema under the *Eberthella lunavensis* organism.

Incidence: Practically never found.

Therapy: Rectal vaccines, subcutaneous vaccines, ectoantigens and the breaking up of bacterial associations.

Achromobacter Pseudocarolinus.

See table for cultural characteristics.

Incidence: Occasionally found. True significance not discernible.

Therapy: No suggestions.

Achromobacter Tardus.

See table for cultural characteristics.

Incidence: Occasionally found. True significance not discernible.

Therapy: No suggestions.

Alcaligenes Bookeri.

This organism is most often found in the dejecta in cases of summer diarrhea of infants. It is a small organism about the size of the *Escherichia coli*, measuring 0.4 to 0.5 μ in breadth and 1.5 to 2 μ in length, short coccobacillary forms being not uncommon. It is non-spore producing. It is a facultative anaërobe and rather easily grown. It has distinct pathogenic action somewhat like the *Proteus vulgaris* but less intense.

Incidence: As mentioned.

Therapy: Since it is rarely found in stools of man, although it may persist in the young adult, it is not of much significance excepting in that connection. It is easily controlled in an antagonistic way with certain strains of *Escherichia coli*, especially those of the Communion grouping. It is also rather easily controlled by proper dietetic measures.

Alcaligenes Fæcaloides.

This organism belongs to the subgenus *Flexnerella* of the partial mannitol fermenters and is best described in the schema under the *Eberthella tangallensis* organism.

Incidence: Occasionally found.

Therapy: Rectal and subcutaneous vaccines, antagonisms usually those of the *Escherichia* grouping, ectoantigens and diet.

Alcaligenes Gintottensis.

This organism belongs to the genus *Lankoides* and is best described in the schema under the *Eberthella pyogenes* organism.

Incidence: Found more frequently in associated infections.

Therapy: Rectal and subcutaneous vaccines, antagonisms usually those of the *Escherichia coli* grouping, ectoantigens and diet.

Alcaligenes Recti.

These occur singly, in pairs and in chains, and appear as rods measuring 0.5 by 1.5 to 2.0 μ .

Incidence: It seems to be a denizen of the normal intestinal canal but can act inimical in associations.

Therapy: Biologic considerations according to these associations.

Alcaligenes Zeylanicus.

See table for cultural characteristics.

Incidence: Uncommonly found.

Therapy: No suggestions.

Bacillus Centrosporus.

This is a thin bacillus with pointed ends, fusiform in shape with slightly granular protoplasm. Usually occurring as single cells measuring 0.4 to 0.5 by 3 to 4.5 μ in dimension. It is a spore producer.

Incidence: Often found.

Therapy: Rectal vaccines, particularly biologic treatments, according to the associations. This organism represents one of a type that produces toxemia from childhood. Chlorine enemas, sera.

Bacillus Flexus.

This bacillus is a large sized organism and a common form found in stools. It measures 1.5 to 1.8 μ in width and 2.25 to 4.5 μ in length, with many bacilli equaling 5.25 μ in length. Usually seen in chain formation, and is a spore producer.

Incidence: Commonly found, and of significance; rarely a straight infection, usually in associations.

Therapy: Controlled by diet, colonic irrigations with chlorine, and rectal instillations according to its associations.

Bacillus Fusus.

These organisms vary in size and shape, are regular in outline with rounded ends, and measure 2.25 to 4.5 μ in length by 0.75 μ in thickness. The longer forms usually predominate. They are spore producers.

Incidence: Commonly found in toxic states. Rarely as straight infections. Usually in associations.

Therapy: Chlorine and other types of destructive bacteria irrigation, and rectal instillations of antagonistic organisms, those mostly found in the *Escherichia* group.

Bacillus Lautus.

This is an active spore producer and is seen as vegetative rods with rounded ends. They vary greatly in size and shape, usually measure $1.5\ \mu$ in thickness by 2.25 to $4.5\ \mu$ in length.

Incidence: Commonly found. Usually of significance in association with other toxic organisms.

Therapy: Destructive medicated irrigations, rectal vaccines, antagonisms usually those of the *Escherichia* group, biologic associations with other organisms important.

Bacillus Mesentericus.

This occurs as rods 0.5 by 1.5 to $3.0\ \mu$ in singles and pairs. They carry spores which are central or eccentric. They belong to the *Subtilis* grouping.

Incidence: Supposed to be non-pathogenic, but of much importance in connection with associated infections.

Therapy: Rectal and subcutaneous vaccines, ectoantigens, antagonisms, *Escherichia coli*.

Bacillus Tritus.

An active sporulator seen in vegetative rods. They present themselves as regular in outline with homogeneous protoplasm and rounded ends. They usually measure 0.75 by $3.75\ \mu$. They occur as short, almost oval, forms.

Incidence: Commonly found, usually in association.

Therapy: Antagonisms, *Pseudomonas* and *Escherichia* groups. Medicated irrigations of value.

Bacteroides Acuminatus.

This organism is frequently found in stools, appearing in pairs with rounded ends, usually arranged like the diphtheria bacillus, that is, in parallel rows.

Incidence: Often found, rarely in a straight-line infection, of most importance in association.

Therapy: Rectal and subcutaneous vaccines, ectoantigens; mostly controlled indirectly by treatment indicated according to the association.

Bacteroides Angulosus.

These occur in rods with rounded ends, singly and in pairs, and are encapsulated. They are anaërobic.

Incidence: Rare, found always in associations.

Therapy: That of the associations, irrigations, séra.

Bacteroides Bullosus.

This is a small rectangular bacillus staining intensely at both poles, occasionally seen with bifurcation at one end.

Incidence: Occasionally found, rarely as a straight infection, and most often in association.

Therapy: Rectal and subcutaneous vaccines. Ectoantigens have been used with benefit. Associations have to be considered in biologic handling.

Bacteroides Cornutus.

This is a small irregular bacillus closely resembling the *Bacillus bifidus*.

Incidence: It may occur as a straight-line infection, but usually in association.

Therapy: Rectal and subcutaneous vaccines, and biologic consideration of the associations.

Bacteroides Dimorphus.

These occur in long rods, granular, singly, in pairs and in chains. It is an anaërobe.

Incidence: Commonly found as straight infections. Sometimes in association.

Therapy: Rectal instillations of antagonisms, those of the *Escherichia* group, vaccines, medicated colonic irrigations, anaërobic serum.

Bacteroides Lævis.

This is a facultative anaërobe, occurring in small slender rods, sometimes singly or in short chains.

Incidence: Occasionally found, usually in associations.

Therapy: Rectal antagonisms, biologic considerations according to the associations, medicated colonic irrigations, anaërobic sera.

Bacteroides Liquefaciens.

These are found in small, slender rods, with rounded ends, usually singly.

Incidence: Often found, sometimes in straight infections, but most often in association.

Therapy: Biologic considerations according to the associations, colonic irrigations with medicament, anaërobic sera.

Bacteroides Pseudoramosus.

This organism resembles the *Bacillus ramosus* of Veillon. It occurs as small delicate bacilli with sharp ends arranged at an angle. It is a strict anaërobe and is indol positive.

Incidence: Frequently found. It may occur as a straight infection, but most commonly of importance in association.

Therapy: Rectal vaccines, but best results are accomplished in breaking up the associations.

Bacteroides Tenuis.

These occur in slender rods, in pairs, and are usually curved.

Incidence: Common. Usually in associated infections.

Therapy: Rectal instillations of antagonisms, biologic considerations of the associations, colonic irrigations with chlorine, anaërobic sera.

Bacteroides Thetaiotaomicron.

This organism appears as a coccus in elliptical form in the shape of a kidney-bean or a bell-clapper, the elliptical forms staining only at the extremities.

Incidence: Commonly found. Of most importance in connection with associations.

Therapy: Rectal and subcutaneous vaccines, and biologic consideration in connection with its associations.

Bacteroides Variabilis.

This is a strict anaërobe of short life duration. As a rule it is very slender and apparently produces skatol.

Incidence: Frequently found. Sometimes in straight infections, but most commonly of importance in association.

Therapy: Rectal and subcutaneous vaccines, and biologic considerations according to the associations.

Bacteroides Variegatus.

These are rods of different forms, sometimes short and regular, sometimes flexuous and filamentous. The ends are sometimes rounded and sometimes pointed. They are arranged in two's, sometimes in the form of a "V." They are indol positive.

Incidence: Commonly found, sometimes in straight infections, and often in associations.

Therapy: Rectal and subcutaneous vaccines, and biologic considerations in breaking up the associations.

Eberthella Alkalescens.

This is a supposedly non-pathogenic organism, morphologically and culturally somewhat like the dysentery bacillus of Flexner. It is distinctly pathogenic in the way of toxin production in the indol series, and significant when present.

Incidence: Uncommonly found, usually as a straight infection, although it may be associated.

Therapy: Direct immunity production by subcutaneous or rectal vaccines and ectoantigen. The associations must be broken up in indicated biologic ways.

Eberthella Ambigua.

This organism resembles the Shiga bacillus, is indol positive and has interest in the work largely in connection with association.

Incidence: It is seldom found, practically never as a straight infection.

Therapy: It seemingly is not controlled by vaccine, and best by attention to incidental associations.

Eberthella Bienstockii.

These occur in short rods, 0.5 by 0.7 μ , generally singly, and are facultative aërobes.

Incidence: Frequently met with. Usually of importance in association.

Therapy: Antagonistic organisms, colonic irrigations with chlorine; biologic considerations according to associated infections and sera.

Eberthella Chylogena.

These occur in rods 0.5 by 1.0 μ , singly and in pairs. It is a facultative aërobe.

Incidence: Commonly found. Sometimes in straight infections and often again in connection with association.

Therapy: Subcutaneous vaccines, ectoantigens, rectal instillations of antagonisms.

Eberthella Dubia.

These occur in rods 0.75 by 2.0 μ , usually singly but sometimes in pairs. It is a facultative aërobe.

Incidence: Occasionally found. Sometimes in straight infections, most often in association.

Therapy: Rectal and subcutaneous vaccines, ectoantigens, biologic consideration according to associations, dietetic and irrigation methods.

Eberthella Enterica.

These occur in rods 0.5 by 1.5 to 3.0 μ , singly, in pairs and in long chains. It is a facultative aërobe.

Incidence: Sometimes occurs as a straight infection; most often in associations.

Therapy: Rectal and subcutaneous vaccines. Biologic considerations to break up the associations. Recently ectoantigens. Sera.

Eberthella Oxygenes.

These are found in rods 0.5 by 2.0 to 3.0 μ , usually singly.

Incidence: Occasionally found. Sometimes in straight infections, but most often in association.

Therapy: Rectal and subcutaneous vaccines, biologic considerations according to the associations.

Eberthella Oxyphila.

These are rods 0.75 by 2.0 μ , usually occurring singly.

Incidence: Frequent. Seen in straight and associated infections.

Therapy: Rectal and subcutaneous vaccines, ectoantigens, biologic considerations to break up associations, colonic irrigations, diet, etc.

Escherichia Alcalescens.

These usually occur in rods, 0.5 by 1.0 to 2.0 μ , singly and occasionally in long chains. This is a facultative aërobe.

Incidence: In straight infections it is practically never seen, and many mistakes have been made in giving it significance in association; however, occasionally it is of importance in this last connection.

Therapy: Controlled by breaking up the associations, usually by subcutaneous or rectal methods. Rectal irrigations of no value.

Escherichia Gastrica.

These occur in rods, 0.5 by 2.0 to 3.0 μ , singly and rarely in short chains.

Incidence: Frequently found. Sometimes in straight infections, not so important in associations.

Therapy: Rectal and subcutaneous vaccines, ectoantigens, antagonisms—*Escherichia*, *Pseudomonas* and *Clostridium welchii*.

Escherichia Iliaca.

These occur in rods, 0.75 by 3.0 to 4.0 μ , usually singly.

Incidence: Commonly found. Most often as straight infections, sometimes in associations.

Therapy: Rectal and subcutaneous vaccines, ectoantigens, and biologic considerations according to the mixture of infection.

Escherichia Neapolitana.

This organism is best described in the schema under the *Escherichia paragrünthali* organism.

Incidence: Very, very seldom found.

Therapy: Rectal and subcutaneous vaccines, the breaking up of inimical biologic associations, diet and irrigations.

Escherichia Pseudocoloides, A and B.

This organism belongs to the genus *Escherichia* and occurs in two varieties, they being best described in the schema under the *Escherichia paragrünthali* organism. The first variety of this organism (A) differs from the *Escherichia pseudocoli* in that it does not ferment dulcitate, and the B variety differs from the *Escherichia pseudocoloides* in that it ferments inositol.

Incidence: Commonly found as a straight and associated infection.

Therapy: Rectal and subcutaneous vaccines, ectoantigens, antagonisms, biologic considerations according to the associations, and diet.

Escherichia Pseudodysenteriae.

These are seen in rods, 0.5 by 1.0 to 2.0 μ , occurring in pairs and in short chains. Pathologic significance not proven.

Incidence: Frequent.

Therapy: Of no special moment. Subcutaneous vaccines.

Micrococcus Ovalis.

This is a common organism of the intestinal canal of infants and adults. It is 0.2 to 0.3 μ in diameter, and 0.6 to 0.7 μ in length. The organisms are generally found in pairs.

Incidence: Frequently found. Usually of importance in connection with association.

Therapy: Controlled by vaccines, rectal and subcutaneous, and according to biologic considerations in control of the associations. Antagonisms.

Pseudomonas Capsulata.

This occurs as small rods with rounded ends, singly and in pairs.

Incidence: Practically never seen in the adult.

Therapy: No experience with it. Seems to be controlled by dietetic measures.

Serratia Havaniensis.

This is a short rod, oval organism, 0.4 by 0.9 μ , occurring singly and in pairs.

Incidence: Commonly found. Pathologic significance not proven, although it may act inimically in associated infections.

Therapy: Biologic considerations according to the associated infection, subcutaneous and rectal vaccines, instillations of antagonisms.

Vibrio Proteus.

These appear as short curved vibrios about 0.4 μ in width and 1.5 to 3 μ in length. They are sometimes seen as distinct spirals.

Incidence: Not uncommonly found; usually as a straight infection.

Therapy: Antagonism. Streptococcus *fæcalis* subcultures, organisms of the *Escherichia* and the *Pseudomonas* growths. Also controlled by medicated irrigations. Difficult of elimination.

The following represent a number of organisms of the Saprophytic group regarding which little is known, and with which the author has not had any experience. No doubt some of these are represented in the above mentioned.

Helicobacterium aërogenes Miller.
Bacillus albuminis Bienstock, 1884.
Bacillus Maidis Cuboni, 1889.
Bacillus subtilis simulans Bienstock, 1890.
Micrococcus leucochromogenes.
Micrococcus oxycyanogenes.
Spirillum roseum.
Micrococcus aërogenes Miller, 1886.
Bacillus intestinalis motilis Sternberg.

THE INTESTINAL ZYMOGENIC ORGANISMS.

Achromobacter Ceylonensis Varieties A and B.

These belong to the genus *Lankoides*, recognition of which is according to the schema under *Eberthella pyogenes*.

Incidence: Practically never found in chronic intestinal infections. The organism has been isolated but three times in five thousand cases.

Therapy: Not much is known.

Aërobacter Aërogenes.

This organism is differentiated from the *Escherichia coli* with some difficulty, and appears to be related to the encapsulated organisms found vegetating on the nasal mucosa. It is widely spread in the dejecta of man and animals. It occurs as a small rod with rounded ends, measuring 0.5 to 0.6 μ in breadth by 0.75 to 1.25 μ in length; is enclosed in a fairly large capsule, which may embrace several individuals. It has no flagella and no spores, is pathogenic to small animals and is a toxin producer in man. The organisms are differentiated from the *Escherichia coli* by peculiar fermentation of sugars and failure to give the methylene-red test, their positive Voges-Proskauer and their ability to overcome acid. They are found more particularly in the upper part of the intestinal canal and less frequently in the lower, and no doubt are an important

factor of toxic states in the small intestines. They are capable of bringing about inflammatory conditions of the bowel, and inflammatory and infective conditions outside of the bowel.

Incidence: Commonly found as a straight and as an associated infection.

Therapy: Rectal and subcutaneous vaccines, ectoantigens, immune sera, biologic considerations according to associations, diet, and occasionally irrigations. Antagonisms.

Aërobacter Diffluens.

This organism belongs to the genus *Proteus* of the type species *Proteus vulgaris* and is best expressed in the schema of the pathogenic organism *Salmonella paradiffuens*.

Incidence: Commonly found in associated infections.

Therapy: Subcutaneous vaccines, breaking up of symbiotic groupings by antagonisms rectally or subcutaneously, ectoantigens.

Bacillus Acidophil-aërogenes.

These were first described by Torrey and are distinguished from the *Bacillus acidophilus* of Moro in that they are found in smaller numbers than these more familiar types. They are widely distributed in Asia and are analogous to the *Lactobacillus acidophilus*. They practically present no more in significance, either in toxin production or in their beneficial use, than the Moro type. The author is in no sense in favor of their use for clinical purposes. However, there is quite an interest in them and the following schema will present the different types known:

L. Acidophil-aërogenes A.

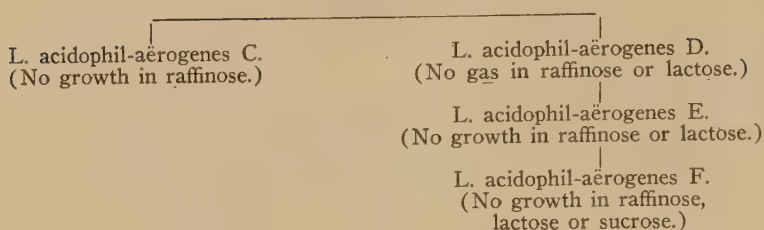
(Gas in all test substances.)

L. acidophil-aërogenes G.
(No gas in lactose.)

L. acidophil-aërogenes H.
(No gas in sucrose.)

L. Acidophil-aërogenes B.

(No gas in raffinose.)



Incidence: A common denizen of the intestinal canal. Made more numerous by lactose feedings. It can be an infecting toxic organism especially in connection with others of the active acid type.

Therapy: Controlled by antagonisms of the *Escherichia coli* grouping, and meat diet.

Clostridium Alcaligenes.

These occur in long, slender rods, singly, in pairs and in short chains. The spores are spherical and polar. The organism is a strict anaërobe.

Incidence: Commonly found, usually in associated infections.

Therapy: Biologic considerations of the associated infection, medicinal irrigations, anaërobic sera.

Clostridium Butyricum.

An anaërobe occurring as rods 0.75 to 1.0 by 3.0 to 10 μ , singly, in pairs or long chains. Spores oval.

A large number of non-pathogenic, motile, anaërobic, butyric acid bacteria have been described, and there are evidently many different strains of anaërobes marked by their capacity to induce butyric acid fermentation. It is difficult to classify these butyric acid bacteria, but since butyric acid formation represents additional volatile acid production they all have significance in toxic gut states. As far as the above-named organism is concerned it occurs as Gram-positive rods, medium in size with rounded ends, singly, in pairs, or in short chains.

It is a spore producer. The organism is less proteolytic than the *Clostridium welchii*. It is non-pathogenic to guinea-pigs.

Incidence: A very common organism in the intestinal canal, and one of the confusing forms as to significance. It does, however, have significance in associated infections.

Therapy: Biologic considerations of these association infections, diet (no fats), and medicated colonic irrigations, antagonisms.

Clostridium Coagulans.

An anaërobe occurring as straight rods, 0.8 by 1.0 to 3.0 μ , with rounded ends and singly, the spores being oval, sub-terminal, and the organisms showing marked bulging.

Incidence: Often found, and of significance in connection with association.

Therapy: Therapy of the biologic consideration of the association, rectal antagonisms, rectal irrigations.

Clostridium Dissolvens.

These occur in slender rods from 2.5 to 12.5 μ in length, usually singly, occasionally in pairs, the spores being oval and terminal. The organism is a strict anaërobe, supposed to be non-pathogenic; however, it has been met with in associated infections, and in my opinion has importance in this connection.

Incidence: Infrequently found.

Therapy: Biologic consideration of the associations of other organisms, anaërobic sera, vaccine instillations into the bowel, and diet.

Clostridium Filamentosum.

These occur as slender rods, singly and in chains, the spores being oval and terminal. The organism is a strict anaërobe.

Incidence: Occasionally found. Usually in associated infections.

Therapy: Biologic considerations for the associated infection, medicated colonic treatments, the use of antagonisms.

Clostridium Filiformis.

These occur as slender rods, 0.5 to 0.8 by 3.0 to 5.0 μ , usually in chains, the spores being small, spherical and subterminal. The organism is a facultative anaërobe.

Incidence: Not infrequently found, and only of significance in association.

Therapy: The therapy of the associations.

Clostridium Fissum.

These occur in long, slender rods, singly or in chains, the spores being small, oval and subterminal. The organism is an anaërobe, having associated significance, especially with some of the aërobic grouping.

Incidence: Infrequently found.

Therapy: Biologic considerations for the associated infection, rectal instillations of strains of *Escherichia coli*, *Pseudomonas*, some strains of *Streptococci* and anaërobic sera.

Clostridium Ovaris.

These occur in straight rods, 0.3 to 0.4 by 6.0 to 8.0 μ , with rounded ends, usually singly or in short chains. The spores are oval, polar, the organism showing distinct bulging at the side. The organism is supposed to be non-pathogenic, and is anaërobic.

Incidence: Occasionally found, usually in association, in which instance its presence in any numbers is important.

Therapy: Biologic considerations of the breaking up of the associations, rectal antagonisms, irrigations, sera.

Clostridium Putrificum.

This organism has already been described under the Pathogenic Group.

Clostridium Regularis.

An anaërobe of large rods, with rounded ends, occurring singly, the spores being large, oval and terminal.

Incidence: This organism is supposed to be non-pathogenic, but when found in association with other toxic organisms has a significance.

Therapy: Biologic considerations of the associations, rectal antagonisms and irrigation treatments.

Clostridium Saccharolyticum.

This is an anaërobe occurring in short rods, with rounded ends, singly or in pairs, the spores being large, oval and terminal.

Incidence: Commonly found, and of significance in connection with associations.

Therapy: Biologic considerations of these associations, colonic irrigations, rectal antagonisms.

Clostridium Welchii.

More popularly this organism is known as the *Bacillus aërogenes capsulatus*, or the "gas bacillus." It occurs as large, encapsulated organisms with round or flat ends, almost never square, usually measuring 0.7 to 0.8 μ in width by 4 to 6 μ in length. As a rule they are arranged singly or pairs end-to-end, and sometimes in short chains. The capsules are regularly present about the organisms especially in the culture forms. They are active spore producers, and are quite strict anaërobes. Much is known about these organisms bacteriologically and they need not be gone into here. It remains to be said that they are definitely pathogenic, and while they occur in the normal intestinal canal usually not more than 2 to 6 to a field, when they occur in larger numbers than these they represent a distinct infection, and produce a true hemotoxin as well as other toxic bodies.

Incidence: Often found, occasionally in straight-line infections, but most often in association with other anaërobes and aërobes of the toxic types.

Therapy: Biologic consideration of the associations of other organisms, anaërobic sera, aërobic antagonisms, instillations into the bowel, and diet.

Clostridium Zoogleicum.

An anaërobe appearing in short rods, singly and in pairs, the spores being large, oval and polar.

Incidence: Frequently found and of significance in connection with its associations.

Therapy: Biologic considerations of these associations, rectal antagonisms, irrigations.

Eberthella Bentottensis.

For cultural characteristics, see table.

Incidence: Frequently found as straight and associated infections.

Therapy: Rectal and subcutaneous vaccines, ectoantigens, antagonisms, and diet.

Eberthella Talavensis.

This organism belongs to the genus Eberthus and is best described in the schema under the Eberthella kandiensis organism.

Incidence: Occasionally found, usually in associated infections.

Therapy: Subcutaneous vaccines, ectoantigens, antagonisms and the breaking up of bacterial associations. Lactose feedings.

Escherichia Acidi-lactici.

This organism differs from the Bacillus lactis aërogenes, is a spore producer, and somewhat represents the same significance as its sister organism.

Incidence: It is often found, and while there is usually a question as to its significance, when present in considerable numbers, it has importance in associated ways.

Therapy: Biologic considerations of the associations, subcutaneous vaccines and ectoantigens.

Escherichia Coli Tropicalis.

These are best described in the Smith's indol-producing division of Durham's non-saccharolytic communis section as

found under the schema in connection with *Escherichia paragrünthali*.

Incidence: Commonly found as straight and associated infections.

Therapy: Rectal and subcutaneous vaccines, ectoantigens, the breaking up of inimical biologic associations, diet and irrigations.

***Escherichia Coloides*, Var. A and B.**

These are best described in the Smith's indol-producing division of Durham's non-saccharolytic communis section as found in connection with the schema under *Escherichia paragrünthali*.

Incidence: Commonly found as straight and associated infections.

Therapy: Rectal and subcutaneous vaccines, ectoantigens, the breaking up of inimical biologic associations, diet and irrigations.

***Escherichia Galactophila*.**

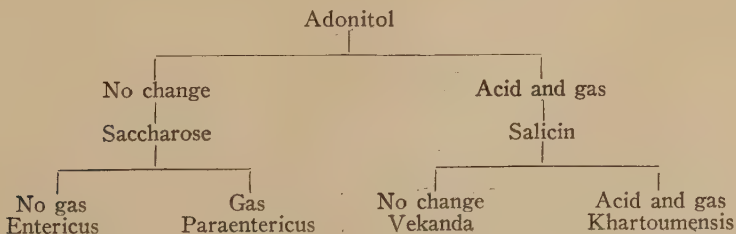
These are found in rods 0.75 by 1.5 μ , usually singly.

Incidence: They become denizens of the intestinal canal, but may present an infection usually of the associated type.

Therapy: Biologic considerations of the association, rectal and subcutaneous vaccines.

***Escherichia Khartoumensis*.**

This organism belongs to the genus *Enteroides*, the type species of which is the *Enteroides entericus* and is best described under the following schema:



Incidence: But rarely found in my experience.

Therapy: No experience with it.

Escherichia Metacoli.

For cultural characteristics, see table.

Incidence: Sometimes found as a straight infection.

Therapy: Subcutaneous vaccines, ectoantigens.

Escherichia Metacoloides.

This organism is best described in the schema found in connection with the pathogenic organism *Escherichia paragrünthali*, and belongs to the Durham's non-saccharolytic communis section.

Incidence: Not infrequently found as a straight or an associated infection.

Therapy: Subcutaneous vaccines, ectoantigens, antagonisms, bacteriologic considerations according to associations.

Escherichia Paracoagulans.

For characteristics, see culture table.

Incidence: Rarely found.

Therapy: Subcutaneous vaccines.

Escherichia Paraentericus.

This organism belongs to the genus *Enteroides* and is best described in the schema found in connection with the organism *Escherichia khartoumensis*.

Incidence: Of significance in connection with straight and associated infections.

Therapy: Rectal and subcutaneous vaccines, ectoantigens, breaking up of biologic associations, antagonisms.

Escherichia Pseudocoli.

This organism belongs to the Smith's indol-producing division of the Durham's saccharolytic communior section and is best described under the schema found in connection with the *Escherichia paragrünthali* organism.

Incidence: Of significance in connection with straight and associated infections.

Therapy: Rectal and subcutaneous vaccines, ectoantigens, the breaking up of inimical biologic associations, diet and irrigations.

Escherichia Pseudocoliformis.

See table for cultural characteristics.

Incidence: Of significance in connection with straight and associated infections.

Therapy: Rectal and subcutaneous vaccines, ectoantigens, breaking up of biologic associations, antagonisms.

Escherichia Pseudocolumbensis.

See table for cultural characteristics.

Incidence: Of significance in connection with straight and associated infections.

Therapy: Rectal and subcutaneous vaccines, breaking up of biologic associations, antagonisms.

Escherichia Schaefferi.

See table for cultural characteristics.

Incidence: Of significance in connection with straight and associated infections.

Therapy: Rectal and subcutaneous vaccines, ectoantigens, breaking up of biologic associations, antagonisms.

Escherichia Vekanda.

This organism belongs to the genus *Enteroides* and is best described in the schema under the *Escherichia khartoumensis* organism.

Incidence: But rarely found.

Therapy: No experience with it.

Escherichia Vesiculosus.

This organism belongs to the genus *Escherichia* and is best described in the schema found under the *Escherichia paragrünthali* organism.

Incidence: But rarely found.

Therapy: Rectal and subcutaneous vaccines, the breaking up of inimical biologic associations, diet and irrigations.

Escherichia Werahensis.

See table for cultural characteristics.

Incidence: Uncommonly found.

Therapy: Subcutaneous vaccines.

Escherichia Wesenbergi.

This organism belongs to the genus *Wesenbergus* and differs from the *Escherichia giumai* in that dulcitol is fermented, both it and the *E. giumai* being indol producers.

Incidence: A distinct pathology and toxin producer. Commonly found as a straight and associated infection.

Therapy: Rectal and subcutaneous vaccines, antagonisms, ectoantigens, biologic considerations according to the associated infections.

Escherichia Wesenbergoides.

See table for cultural characteristics.

Incidence: Has a distinct pathology and is a toxin producer. Commonly found as a straight and associated infection.

Therapy: Rectal and subcutaneous vaccines, antagonisms, ectoantigens and biologic considerations according to the associated infections.

Lactobacillus Acidophilus.

These are branching organisms occurring as rods, and are found mostly in the stools of infants. They occur in a number of different varieties, as mentioned below, and usually are considered as non-pathogenic. There is much literature on them because they have been used extensively for the treatment of intestinal toxemia. In the intestinal canal, where they are viable if taken in large enough numbers, they render the contents of the gut acid and in that sense somewhat hold back a status of putrefaction, but they cure nothing. Suspension of their use usually causes a complete resumption of the toxic

status, but not a few of the really pathogenic toxic states progress with these organisms in which they act in association in intensifying the toxemia, especially in the acid producing bacteria. Their use by mouth in a therapeutic way represents one of the fallacies in medicine today. Since there are a number of different strains they have been described by Rahe as follows:

L. Acidophil-aërogenes.

Type	Maltose	Dextrose	Lactose	Sucrose	Raffinose
A	Gas	Gas	Gas	Gas	Gas
B	Gas	Gas	Gas	Gas	Acid
C	Gas	Gas	Gas	Gas	—
D	Gas	Gas	Acid	Gas	Acid
E	Gas	Gas	—	Gas	—
F	Gas	Gas	—	—	—
G	Gas	Gas	Acid	Gas	Gas
H	Gas	Gas	Gas	Acid	Gas

Incidence: A common denizen of the intestinal canal. Made more numerous by lactose feedings. Since they often indirectly produce toxemia they are not mentioned in the non-pathogenic organisms.

Therapy: Controlled by antagonisms of the *Escherichia coli* grouping.

Proteus Asiaticus.

This organism belongs to the Asiaticus division (indol formers) of the Paratyphoid-Asiaticus group of the type species *Salmonella paratyphi*, genus *Salmonella*, and is best described in the schema of the *Salmonella mobilis* organism.

Incidence: Not uncommonly found in straight and associated infections.

Therapy: Rectal and subcutaneous vaccines, the breaking up of biologic bacterial associations, and dieting.

Salmonella Archibaldi.

This organism belongs to the genus *Salmonella* in the Asiaticus division of indol formers and is best expressed in the

schema of the pathogenic organism *Salmonella mobilis*, to which the reader is referred.

Incidence: Practically never found here.

Therapy: Nothing offered.

Salmonella Giumai.

This belongs to the genus *Wesenbergus* of the type species *Wesenbergus wesenbergi*, differing from the *Wesenbergi* in that dulcitol is not fermented.

Incidence: Not infrequently found in straight infections.

Therapy: Rectal and subcutaneous vaccines, ectoantigens, breaking up of inimical associations and antagonisms.

Salmonella Morgani.

For cultural characteristics, see table.

Incidence: Occasionally found in dysenteric patients.

Therapy: That of the acute condition.

Salmonella Veboda.

This organism belongs to the *Veboda* group best described in the schema under the *Salmonella willegoda* organism.

Incidence: Not infrequently found as a straight or as an associated infection.

Therapy: Subcutaneous and rectal vaccines, ectoantigens, biologic considerations in breaking up symbiotic cliques.

Salmonella Watareka.

For cultural characteristics, see table.

Incidence: Sometimes found as a straight and associated infection.

Therapy: Subcutaneous vaccines, ectoantigens and biologic considerations according to the associated infections.

Salmonella Woliniæ.

This organism belongs to the *Paratyphoid-asiaticus* group best described in the schema of the *Salmonella mobilis* organism.

Incidence: Practically never found.

Therapy: Nothing to be offered.

Regarding the following organisms, little is known. They probably represent organisms already mentioned:

Bacillus of Miller.

Bacillus lactis aërogenes Abelous.

THE INTESTINAL SPIROCHETES.

The spirochetes which occur in the intestines and which may cause toxic states are *Spirochæta eurygyrata*, the *S. stenotrepta* (Werner), and the *Emendavit* (Fantham). These are often met with in stools, sometimes most numerous. To me they are toxic infection organisms requiring attention. As a rule they are associated with various bacterial organisms of infective combinations, these requiring individual attention. While most of the Vincent's infections in the bowel are quickly fatal, a chronic form with ulceration may be met with. For the control of spirochetes in the intestines, solutions of arspenamine (5-8 mg. per kilo weight) or neoarsphenamine (13 mg. per kilo weight) are required by transduodenal lavage or by colonic irrigations. Small doses of arsenic preparations taken by mouth over long periods of time have also been helpful.

THE INTESTINAL NON-PATHOGENIC ORGANISMS.

Bacillus Subtilis.

This was originally described as the *Vibrio subtilis*. It occurs as a small, homogeneous bacillus, measuring 0.4 by 1.5 to 2.5 μ in twenty-four hour agar cultures. The spores arise in the center or toward one end of the rods and are slightly greater in diameter than the rods, thus causing distinct bulging. The free spores, when split off, may retain bits of protoplasm at each end.

Incidence: Occasionally found, and only of significance in associated infections.

Therapy: Biologic considerations of these associated infections, chlorine enemas.

Bacteroides Bifidus.

This organism was originally described by Tissier as present in the stools of infants, and has been described at length. It appears as a slender bacillus in length from about 2 to 3 to 5 to 6 μ , with an average length of 4 μ . It is usually arranged as a diplobacillus with proximal ends swollen, and the distal ends tapering or pointed, and separated by unstained areas. These diplobacilli usually lie side by side in parallel bands, sometimes showing the true branching as "Y"-shaped forms. It is non-sporulating.

Incidence: Ordinarily it is described as non-pathogenic, but in my opinion it is pathogenic in some instances, especially in some associations. In its significance it is about the same as the *Lactobacillus acidophilus*, although as a toxic organism it seems to be more significant. It is a strict anaërobe. High infection may persist from infancy through adult years. It is when the latter occurs that it is important.

Therapy: Biologic considerations to its associations, diet negative in milk products and colonic irrigations of alkaline solutions.

Bacteroides Fragilis.

This occurs as small, short, non-motile rods, with rounded ends, singly or in pairs, looking like a diplococcus. It is distinctly pathogenic to guinea-pigs and rabbits, and in my opinion is definitely capable of producing toxic states in man. However, it is usually classified as non-pathogenic.

Bacteroides Oviformis.

These occur as a short rod or an elongated coccus, or more often as a diplococcus made up of oval elements, resembling the ordinary form of the enterococcus. It is a strict anaërobe, and in all probability non-pathogenic.

Incidence: Not important.

Therapy: Not important.

Bacteroides Ramosus.

This is a small, slender bacillus, usually occurring singly or in pairs. It is considered in the non-pathogenic grouping, although I believe it once in a while has distinct significance in the intestinal canal of man.

Incidence: Of importance in connection with associated infections.

Therapy: Subcutaneous and rectal vaccines, biologic considerations of the associations, rectal antagonisms largely in the *Escherichia* groups.

The unknown non-pathogenic organisms, which are probably duplicated in the before-mentioned, are the following:

Bacillus satellitis.

Bacillus anaërobis liquefaciens Sternberg.

Addenda.—The Saprophytes, *Bacillus megatherium* and *Bacillus mycoides* were omitted in their positions in the description.

Bacillus Megatherium.

This is the largest of the spore producing organisms; usually 0.75 to 1.25 by 6 to 9 μ . On culture they may measure 30 to 40 μ in length. The spores are central or toward one end, and there may be two spores to the one bacillus. They are commonly found in the intestine, usually in associated infections, and are controlled with difficulty.

Therapy: Control of associated infections; frequent bowel eliminations, enemata (colonic irrigations) of chlorine solutions.

Bacillus Mycoides.

Found in milk, water, dust, etc. The organism has square ends, 0.5 by 3 to 6 μ in size, with central or one end spores. It is a common denizen of the intestinal canal, significant in single and associated infections.

Therapy: That of the associations, enemata (colonic irrigations) of permanganate of potash or chlorine solutions, and small doses of calomel by mouth. The infection is difficult to control.

CHARTS

1. Cultural Characteristics of the Intestinal Pathogenic Organisms.
2. Cultural Characteristics of the Intestinal Saprophytic Organisms.
3. Cultural Characteristics of the Intestinal Zymogenic Organisms.
4. Cultural Characteristics of the Intestinal Non-pathogenic Organisms.

The above mentioned Charts will be found inserted immediately following the Index at the back of the book.

CHAPTER XI.

BIOLOGY—BACTERIAL TREATMENTS.

IMPORTANCE OF BIOLOGY.

Medicine in its interpretation is mostly based on biology. Biology in this connection, as in others of significance, is a field of interest that will be found relative no matter how far back one goes. Depending upon the character of the deductions, it can be considered all the way from a "mess" that starts nowhere and gets nowhere to a fabrication of worthwhile interest and endeavor. Depending upon one's personal attitude, one would base judgments upon a too optimistic attitude, or upon one of negation and depreciation (such as I had at the beginning and held for years) until convinced from the results accomplished that the biological application of this work is a most valuable contribution to medicine and worthy of a life's study and interest.

It must be evident that biology has a long way to go before it possesses even a few of the fundamentals of a science. In its application to medicine for the human being it is confused on every side with the relative influences of heredity, racial types, age, environments, physiology, psychology, immunities, morphology, etc. These in themselves make deductions difficult enough, but when to them are added the great difficulties of biologic diagnosis in chronic toxic intestinal disorders and the still greater difficulties of biologic treatment, the field of work requires not only the foundations of high ability in diagnosis and internal medicine, and at least apt diagnostic knowledge of the specialties, but a vast experience in the biologic field also. I doubt that, short of years of rather constant work clinically and biologically, a person can acquire better than a fair degree of ability in intestinal work, and the hard part of this is that both sides must be done by one person, or two persons in most close association.

The main reason for this is that biology in medicine, as stated, is a relative matter and all related sides must be judged by the one mind. If the analyses are made by one person (such as a laboratory worker) and then interpreted by another (such as the clinician), the personal equations, the unfixed fundamentals and ever-shifting phases of medicine cause the relationships and significance to be lost or so badly obscured that as a rule neither one gets anywhere. Generally, unless all of the work is done by one person, and not entered into practically until several years of experience have been acquired, I strongly recommend that it be not engaged in at all. Firstly, because the patient would largely be exploited, and this is not fair to the public or this field of work, and secondly, opprobrium and discouragements from ignorance would be the inevitable result. To go into it for commercial and "making-a-living" reason is as unpardonable in the first instance, as it will quite likely prove a poverty existence in the second.

LOCAL AND TISSUE IMMUNITY.

Natural or "innate" immunity is that which protects normal animals against slight or moderately virulent infectious agents, and may in general be largely ascribed to the polymorphonuclear leukocytes of the circulating blood. Acquired immunity, whether expressed actively in a recovered or actively immunized animal, or passively through transfer of the serum of hyperimmunized animals, involves certain new and enhanced physiological activities. These activities are the antagonistic substances usually designated as antibodies. There are no doubt many more immunizing substances produced that would be successful against the streptococci, staphylococci and various forms of intestinal infections acute in nature, and also the highly specific, such as the Shiga bacillus of dysentery, typhoid fever and cholera.

It is probably also true that the reticulo-endothelial system of cells is particularly concerned in active phagocytic opposition to infectious agents. Certain compounds of this system are proven by various dye tests to be distinctly concerned in the process.

By enteric fevers are commonly meant those conditions that are caused by the *Eberthella typhi* and *Salmonella* paratyphoid organisms. In addition to these, conditions resembling enteric fever are found in those of the parenteric class in which are prominent the organisms of the *Eberth* and *Alcaligenes*, the genus *Salmonella*, genus *Enteroides*, genus *Lankoides*, genus *Balkanella*, genus *Wesenbergus*, and the genus *Escherichia*. These latter types of acute enteric disturbances are generally not diagnosed in their true etiology, but are classed as enteric fevers, and most often presented as due to the organisms of the paratyphoid A or B classes.

In this volume we are not concerned with acute or diarrheic states, but solely with chronic infections and their toxemias, yet it may be stated as a biologic truism that practically all the organisms which can cause an acute condition can also cause a chronic one, and very commonly a chronic one without an acute initial onset. Although many a chronic toxemic state does begin with an acute diarrhea, this usually is of such short duration that it escapes observation or attention.

THE UTILIZATION OF CARBOHYDRATES BY BACTERIA AND BACTERIAL PRODUCTS.

Changes in Bacteria-food Products.—To no one more than A. I. Kendall belongs the credit of having drawn attention to the fact that, as a rule, organisms utilize carbohydrates in culture media before proteins. This is a most important matter to keep in mind for the understanding of intestinal conditions of a biologic nature and has a considerable significance in connection with the dietetic handling of these conditions. This matter is of no small importance because often, depending upon the character of the carbohydrates, substances are produced by bacterial action, all of which are resorbable into the general body.

The *proteus bacillus* produces large amounts of indol, and forms a soluble, proteolytic enzyme when it is cultivated in a suitable nitrogenous medium. But if, before the organism is cultivated, some glucose is added to the nitrogenous medium, it no longer produces indol nor the proteolytic enzyme, but

forms considerable amounts of lactic acid instead. In a similar manner various strains of the *Escherichia coli* produce indol and phenolic bodies from the nitrogenous constituents of ordinary nutrient broth and food, but the addition of glucose will change the resulting substances into a lactic-acid-producing microbe. Thus the addition of utilizable carbohydrates to nitrogenous cultural media in which bacteria are growing affects the character of the products they form very strikingly. Kendall has shown that, in general, it may be said that utilizable carbohydrates protect the protein from direct bacterial utilization for energy.

On the other hand, it must not be forgotten that, under certain circumstances, the addition of utilizable carbohydrates may indirectly bring about some alteration in the protein constituents of cultural media as well as the intestinal canal which might not otherwise take place. Utilizable carbohydrate in connection with the *Clostridium welchii* indirectly leads to the appearance of a histamine-like substance, which is not produced when carbohydrate is absent. Among the various substances which are formed in carbohydrate utilization by bacteria are butyric acid, glycerol in which acid is produced from the triatomic alcohol, acetic, lactic, propionic, oxalic acids, etc. Among those produced in protein cleavage are acetone, ammonia, methyl guanidin, cholin, neurin, putrecin, cadaverin, mercaptan, hydrogen sulphide, phenol, cresol, skatol, indol, etc.

Various organisms bring about the formation of products characteristic of them, and the activities of the different types—and indeed most types of bacteria—are somewhat determined by the nature of food upon which they subsist. If the food contains utilizable nitrogenous substances and none or but very little available carbohydrate, intensive nitrogenous metabolism takes place, with the formation of ammonia, amino-acids (including tryptophane), amines, indol, etc. In the presence of fermentable carbohydrate, however, the metabolism assumes a different aspect and becomes essentially fermentative. Since practically all organisms in the intestines are facultative, persistency with diet does not long carry out modification of toxin

production, certainly not longer practically than up to thirty days. This fact seems to be difficult for the profession to learn.

Let me state this briefly and candidly: a diet may change the biology and chemistry for a short period, but in a short time the facultative abilities of intestinal organisms make them able to multiply, and the infection and toxemia are again present, no matter how opposite the diet may be. The sooner the internist, gastro-enterologist, the food faddists, food types of sanitaria and the public realize this, the sooner good work in intestinal toxemia will be accomplished.

Bacterial Toxins.—Also important with the modifications which diet can bring about in the known chemical bacteria-food products are those generated from the bacteria themselves—the bacterial toxins. These essentially are products identical, irrespective of any types of foodstuffs the bacteria happen to be existing and multiplying on. The main shortages of dietetic toxemia work have been in lack of consideration of these. Efforts and interests have been engaged in regard to diet and chemical matters pertaining to the bacterial food products, but no one has been interested especially in the bacteria and the associations present, and the bacterial toxic products therefrom. Interest in the latter is accepted in diseases like diphtheria, anthrax, blood infections, etc., but in the intestinal toxemias nobody has thought about them, even though they are the major factor in condition and disease production. It will be a long time before chemistry will be abreast with methods of examination pertaining to them. But meanwhile the clinical studies of the human being in his disorders and diseases show that these toxic substances are important matters, and since there are methods for study of the bacteria themselves, an interest in this part of the study answers as well now as it will in future years when such chemical matters have been devised. Because, when they have been devised, the use of such methods of examination would always be indirect as compared to the examinations for the identification of the bacteria themselves.

The so-called Voges-Proskauer reaction takes account of those products of fermentation produced by some of the microbes, not brought about by the fermentation of simple

sugars, with or without gas production. There have been found with certain spore-producing organisms, such as the *B. subtilis*, *B. mesentericus* and *B. aërobacter aërogenes*, which are more or less spore-forming bacteria, and they produce the color reaction elicited in glucose cultures by the addition of enough caustic potash solution to render the reaction of the medium very alkaline. This reaction is brought about by some unknown constituent of peptone in the presence of caustic potash.

ANAËROBIOSIS.

Anaërobes are distinguished from aërobes in that the former are able to live in complete or nearly complete absence of atmospheric oxygen, and usually require elimination of free oxygen from their environment for growth and multiplication. Organisms are distinguished in three groups: obligate anaërobes, obligate aërobes, and facultative aërobe-anaërobes. The best-known obligate anaërobes are found among the sporulating bacilli. Pasteur's explanation of anaërobiosis was that the organisms secure their oxygen through the fermentation of carbohydrates. The anaërobes of the greatest significance in connection with intestinal work are the *Clostridium welchii*, *Clostridium butyricus*, *Clostridium regularis*, *Clostridium tetani*, and *Clostridium putrificus*.

It is by no means certain that obligate anaërobes require an absolute exclusion of oxygen for their maximum growth, although the amount necessary to inhibit growth is very small. The physico-chemical conception holds that obligate anaërobes differ from the aërobes by the fact that small amounts of oxygen are inhibitive to their growth and toxic to their protoplasm.

An interesting phenomenon in connection with anaërobic growth is the aërobe-anaërobe symbiosis in which certain anaërobic organisms will grow best in association with certain aërobic forms. The aërobic organisms of the intestinal canal which have a stimulating effect upon anaërobic growth are particularly the *Erythrobacillus prodigiosus*, *Proteus vulgaris*, *Pseudomonas aëruginea* and the *Escherichia coli* group. Why

this is true still remains an unsolved problem, but it is true, and its great importance in connection with intestinal toxemia must ever be kept in mind in the estimation of toxin-producing combinations in which aërobes and anaërobes are in association.

ANTIGENIC PROPERTIES OF THE BACTERIAL CELL AND ANTIBODY REACTIONS.

The processes of immunity by which the animal body resists invasion and injury by bacteria are merely specific instances of the operation of general biological laws applicable to a field far wider than that of infectious disease. When we speak of immunity, we speak of a physiological response, which, in its broad biological significance, may be included under various divisions. Perhaps with lipoids and carbohydrates not much need be said in the matter of the resistance met with in normal metabolism, but with complex nitrogenous material, proteins or closely related substances, this is not the case. Here contact with the tissue cells sets in motion an emergency mechanism which reveals itself by a specifically changed reaction capacity of the tissue cells.

It is important, however, in clinical medicine to distinguish between the effects of foreign materials—as to whether they call for antibody formation or not. This is a matter which has to do entirely with the presence of an antigen in the introducing substance. Antibody formation is a regular response to the parenteral administration of coagulable proteins and cellular antigens. Bacterial bodies contain a variety of biologically active materials, some of which are truly antibody-inducing substances. In infectious disease we have a good instance in which this physiological process is set in motion.

All immunity reactions, both those between the antigen and the cell, as well as those between the antigen and its antibody, must be dependent upon chemical affinities. When an animal is infected with living bacteria, the processes set in motion are complex and by no means completely understood, but there is no doubt that they are proven to be present by the study of infectious disease. When the animal is infected with living

bacteria, or when dead bacteria are administered without any extensive solution of the bacterial bodies, these antigens arouse the formation of specific antibodies with effects which we are familiar with as agglutination, precipitation, opsonization, etc. effects which are believed to be due to a single sensitizing anti-substance. Most responses to bacterial antigens are specific, and were they believed not to be specific, there would be no occasion for the writing of this volume. These specific substances are soluble, becoming residue substances probably retained mostly by the protein. These antibodies assist in increasing the ability of the body to get rid of foreign antigenic substances by causing lysis, in all cases phagocytosis, and no doubt certain chemical resisting substances.

BACTERIAL SYNERGISM.

Coupled Reactions.—A number of years ago Castellani and Taylor called attention to a phenomenon, which, while it had undoubtedly been noticed before, had not received the attention it deserved. This has recently been described by Fiallos as follows: "Two bacilli, neither of which causes the production of gas in certain compounds, may do so when artificially mixed together, provided one of them is capable of producing simple acidity (never gas) in these compounds, and the other, though inert to these compounds (*i. e.*, produces neither acid nor gas), is capable of producing gas from glucose."

This phenomenon is due to a coupled reaction, and is best illustrated between a staphylococcus and the *Salmonella Schotmülleri*, which when acting together produce acid and gas from nutrient lactose-fermentation media, although the staphylococcus alone merely produces acid, but no gas from this sugar, and the *Salmonella Schotmülleri* fails to utilize the sugar at all. Many examples of this, in varying degrees, are found in the intestinal canal in association with different bacteria that are met with there. Another type of coupled reaction is that described by Kendall in relation to gas formation in milk by *Escherichia coli* and an associated actively proteolytic organism—for example, the *B. mesentericus* which does not ferment

lactose, but forms gas in considerable amounts. The coupled reaction is termed synergism.

THE SIGNIFICANCE OF THE AGGLUTININS.

Much thought has been given to the functional rôle of agglutinins in infectious disease, that is, the part agglutination as such plays as a factor in protecting the body against infection. The agglutinative property of the body fluids of both normal and immune animals is exercised upon many bacteria, and there are no diagnostic tests more satisfactory today to show that the body is resisting in some specific direction. In this type of diagnostic work, when one knows what the organism employed is that produces a marked agglutinating reaction, one assumes that this organism has to do with the infection at the time.

As has been explained elsewhere, diagnostic agglutination work in estimating bacterial infections in the intestinal canal is of very limited value. While the agglutination reactions may serve to useful purpose in infections elsewhere in the body, my experience with it in intestinal diagnoses is that it fails more times than it is useful, and leads to too many errors of bacteriological significance to be of any value as a procedure in any routine sense. When after all the clinical, x-ray, technical laboratory work on feces, urine, blood, etc., has been done and an intestinal biologic error proven, occasionally these tests of the blood serum may help to serve the purpose of unravelling a symbiosis. But in identifying a bacterial condition in direct infective ways, unfortunately these tests are almost useless. Another reason for the failure is that the body has a number of agglutinins naturally, and there are so many organisms in the intestinal canal, that to assume a significance in this or that way usually leads to diagnostic, clinical and therapeutic error. This factor of error is so high that the agglutinating reactions are no longer depended upon in my work, are performed but rarely, and then only with the hope of getting some additional data in complex biologic situations.

Here and there in straight infections it has been found that the degree of agglutination and opsonization of bacteria outside the body is in general inversely proportional to the virulence of the non-significant microorganisms. Agglutinins, opsonins and phagocytes are very closely related, and no doubt exert an important rôle in resistance to and recovery from infection.

PRECIPITINS.

Following the demonstration of antibodies causing the specific clumping or agglutination of cellular antigens, came the description of antibodies causing precipitation of unformed protein materials employed as clear solutions. There has been suggested in this connection that each antiserum reacted for the most part only with the homologous antigen. It must be assumed that the reaction leading to the appearance of the visible precipitate, the ring or the flakes, is closely similar to those initiating agglutination of cellular antigens. The properties of precipitins are like those of the agglutinins. They are fairly heat-resistant antibodies, present for the most part in the serum of immune animals only, and their activity does not depend upon the presence of complement.

The manner in which precipitins act upon homologous antigens is not clearly understood. Many observers have used various tests with the presence of bacterial precipitinogen, but the information obtained, while perhaps useful in certain single line infections locked up in various parts of the body, has been of no value in intestinal work of chronic biologic types.

ORIGIN OF ANTIBODIES.

An antibody is elicited from an antigen that happens to be introduced and usually possesses the power of reacting specifically with such antigen, destroying or modifying its action or neutralizing its poison. It is believed that they are protein in nature or closely attached to one or another protein. It is probable that the spleen, and secondarily the lymph nodes, bone marrow, liver, kidney, lung and omentum, are involved in the production of antibodies since they are present in some or all

of these organs before appearing in the serum. It seems that the formation of antibodies may depend on some element common to all these organs, such as the lymphoid tissue, hemato-poietic tissue, or reticulo-endothelial cells. It is probable also that the extracts of various glands are concerned in antibody formation, because it is proven that the adrenals have an enhancing power either in the production or in the activity of antibodies to kill bacteria.

Most observers believe that no one organ in the body can be responsible for so important a function as antibody production, and they consider that many or all the cells in the body can and do contribute to immunity response. Since there is such a widespread distribution of lymphatic tissue, hemato-poietic tissue and reticulo-endothelial tissue, this belief in antibody production is probably correct. The tissue of the phagocytes, of course, is most actively involved in the production of antibodies.

THE BACTERIOPHAGE.

According to D'Herelle, transmissible lysis is the result of an acute infectious disease of bacteria. This is believed to be so because in filtrates of cultures subjected to the action of bacteriophage and examined under an ultra-microscope, the presence of minute granules is noted. According to D'Herelle some of these granules can be seen entering the bacterial cells and multiplying within them. The bacteriophagic substance is a lytic principle present in the medium in a state of true solution, formed by separate autonomous units of an active agent. Thus the filtrate of lysed cultures is purified by removal or digestion of bacterial protein, leaving only the active substance, and the antisera obtained by immunization with such purified preparation do not react with bacteria while they do neutralize the phage.

Moreover, in instances of phages capable of reacting on several related species of bacteria, the antigenic specificity of filtrate is constant for each phage and is independent of the bacterial substratum which was used to obtain the active filtrate. I am among those that agree that although bacterio-

phages may act against different species of bacteria, there is not a single bacteriophage common to both man and animal capable by adoption of acquiring a virulence against all bacterial species.

The fact that accumulation of active agents in the medium is independent of lysis, but is intimately connected with the active growth of bacteria, strengthens the view expressed that the phage may be some product of bacterial metabolism.

The intestinal canal of the fetus does not contain any bacteriophage. In the first days of extra-uterine life, however, the intestinal canal becomes infected with bacteria, and some time, about the fourth to the seventh day after birth, phage can be demonstrated in the feces. Usually this phage is active against *Escherichia coli* only.

Although D'Herelle and others have written on the subject of the bacteriophagic methods of treating intestinal conditions, laboratory and clinical experience does not sustain the views advanced by these workers as a means of treating biologic intestinal toxemias. The hope of using bacteriophage as an agent for prevention and therapy in infection, lies in finding the means of removing the obstacles of infectivity in its natural environment, thereby allowing the bacteriophagic substance to act as freely as it does in a test-tube, a manifestly impractical application to clinical medicine, and especially to the intestinal canal. On the other hand there is no doubt that the bacteriophagic methods can be used in mixed bacteriologies of feces in subcultures in the study and examination for the purpose of coming down to the identification of one or more groups that will persist in the end.

Not uncommonly the organisms which persist are important etiologic factors and indices for the treatment of this or that individual case. This, however, cannot be taken as generally true in the biologic diagnosis, because often it fails as a method. It may be the important lead here and there, it may have collateral value, or be an item among others which would be significant, or it may not help at all, and even lead to mistakes. Only ample studies and experience in this work could be helpful in judgments of this method of examination.

While almost invariably the bacteriophagic methods have served to no value in the work, and the results are in no sense to be compared with the other biologic methods mentioned, here and there benefit has been noted. Having no faith in the value of taking the phagic substances by mouth in this line of work, the phagic substances have always been introduced by rectum, and rarely intravenously. The difficulties of evaluating this type of treatment with any degree of accuracy are insurmountable, and since it is too questionable a proposition and because of the many known differences in bacteria and their strains, the phages of which are still unknown, the whole work should be considered as "loose" in a scientific sense and too much of a "hit and miss" affair. My experiences with it may be summed up as follows:

There have been no acute cases of dysentery handled, but in several of the chronic forms of infection of the true dysentery organisms, benefit has come, but not to the extent accomplishable with the judicious use of the biologic methods mentioned. Failures were quite uniform in chronic colitis, the same being true in chronic typhoid and the paratyphoid infections. If very careful work in bacterial diagnosis is done in the *Escherichia coli* infections and definite phage to the bacterial strain or strains is made, benefit had been accomplished here and there. As a method it is quite worth the while in high staphylococcic content in the bowel, and at times has my distinct preference. In streptococcic infections of the intestine it is useless. Throughout the many organisms in my work it represents the last method to be employed in the definite causative ones. The treatments are simple once the laboratory work has been done, and not infrequently it serves to purpose in attempts to clear off incidental forms or the breaking up of symbiotic cliques.

Of course, phagic tests must be made with whatever organisms are selected for attack before treatments are established. This is essential because one can never be sure that phagic factors are associated with any of them, the intestinal organisms largely not having been studied in bacteriophagic ways. In this same connection with the less well-known in-

testinal organisms quite striking phage production is often met with, which, according to the case, may be valuable in treatment when associated with the other methods of more direct, specific and scientific attack against those more likely to be causing the infection.

ANAPHYLAXIS AND ANAPHYLACTOID REACTIONS.

Hypersensitiveness to various foods has been long known, and the word "allergy" has been offered as a comprehensive term covering the various manifestations, more particularly in man. Of late years the term "anaphylaxis" has come to have a special meaning, and the term should be restricted to the condition of hypersensitivity to definitely antigenic substances. Much work has been done by various observers in clinical medicine in the study of allergy in connection with various foods.

For several years various types of dermal tests have been employed with the different food proteins as a basis upon which to standardize a diet. There is no question that here and there clinical results worth while are deducible from the tests. Many times, however, the best results are obtained from the patient's history through fasting from various food substances which have produced anaphylactic phenomena. As a rule these individuals have learned not to eat these foods. The additional data on foods particularly that has come from the study of the various dermal tests has in my experience been almost worthless.

I know there are not a few good observers in medicine who employ this method of standardizing a diet, and to them I would leave this thought. There are a few individuals who are sensitive to certain types of food, but, in the majority who are anaphylactic, the significance and the importance of this or that food cannot be judged by dermal reaction, and when certain definite reactions do occur one is liable to be more in error in advice to the patient than if one had not done the dermal tests at all. The reason for this is because food substances gaining entrance into the intestinal canal and going through the process of digestion are no longer the

substances as introduced, and the intermediate and end products are usually no longer characteristic of the food. On top of this is the most important part of this subject of food allergy, namely, that the biology and bacteriology of the intestinal canal is not taken into consideration when making the skin allergy food tests. There are not a few individuals who are distinctly allergic, not because of certain foods that they would eat, but because of the biology of the intestinal canal, and unless one makes investigations of the intestinal canal at the same time, the whole work in allergy both diagnostically in the dermal tests and therapeutically is incomplete and unsatisfactory. Thus there are innumerable individuals who are allergic because of the biological conditions in the intestinal canal and not only because of this or that food that is eaten.

The reason allergic studies have largely fallen down is that these facts are not taken into consideration, and it is not until the medical profession does so, that the importance of allergy will be understood. Finally, biologically produced allergies are factors of the greatest significance in connection with not only local conditions in the abdomen, but many diseased and disordered states of the rest of the body.

It might finally be mentioned that very largely in my work various anaphylactic reactions used in biologic methods of treatment by rectum occur in individuals. Except in subcutaneous administrations, these are never manifested in the usual skin reactions seen in serum sickness, etc., and are invariably of a vague type. The most common is a local reaction in the colon consisting of an acute attack of gas distention and various degrees of abdominal distress. Another is an overpowering lassitude in which the individual may desire to sleep for two or three days, or just "feel tired all the time." Another, rarely seen, is certain mental phenomena in which individuals are capable of going about and conducting themselves in the proper manner and perhaps are not at all aware of where they are, who they are, or where they live, this lasting for a short time. All such and more minor types of reactions are invariably of short duration, usually last but a few

hours, never as long as a day, and are almost always terminated by one or two moderate-sized doses of acetyl salicylic acid. When they occur, they should be judged as evidences of immunity, and generally call for cessation or modification of bacterial treatments.

The hypersensitization then usually wears off in the course of time, and if the biologic examinations suggest that an infection is not controlled, more or less short runs of bacterial treatments may again be called for and safely conducted. The majority of the patients have no marked anaphylactic reactions, neither local in the colon nor general in the body. Those mentioned above are the extreme instances and are not met with oftener than five cases in one hundred. Perhaps in eighty out of a hundred slight phenomena are met with. In some these reactions from rectal instillations occur at the beginning of treatment and not at all after the first month or two. In others they occur at any time, most commonly near the end of treatment. The symptoms may be severe or they may be most trivial, and this is generally but not totally true whatever the bacteria may be or the combinations. There is no rule or order in their occurrence. Since with most all of the bacteria they occur at some time and in degrees, and commonly take place at the time of the establishment of immunities, they are looked upon as of good omen.

It is my opinion that they are often due to the flooding of the body with antibody and resisting substances, rather than due to the specific protein or the toxins of the instillations themselves. In states of sensitization, however, the introduced protein and toxins of the bacteria can act as additional factors. Judgments as to modification, amplification or cessation of doses require familiarity with these reactions, the organisms being used, and the results of check-up of stool examinations. Occasionally shifts from bacterial vaccines to the ectoantigens from the same bacteria will modify the reaction somewhat, and this is sometimes in order in severe reactions without high degrees of immunity. No attention is paid to slight malaise.

What is stated above pertains particularly to mental phenomena, more or less collapse types of reactions, and to minor

grades and degrees. Not a few cases accomplishing perfect immunities have no reactions whatever all the way through the treatment. As a rule where there are no reactions and the check-ups of the stools show no accomplishments, either the biologic diagnosis or the methods are wrong, or the case is the rare one met with in which worth-while results cannot be brought about. The occasional instance of such conditions always requires judgment and study, reëxamination, changing of methods, or the stopping of the treatments.

VACCINES IN THE TREATMENT OF INTESTINAL CONDITIONS.

It is a well-known fact that after the production of disease by bacterial invasion, a certain degree of immunity exists for some time. Usually this is relative and but seldom absolute. When Edward Jenner observed and showed experimentally (1796) that when cowpox virus was inoculated into the human being a trivial infection called vaccinia was induced, and that this was followed by an absolute or nearly absolute immunity against smallpox, and when Pasteur laid down the basis of scientific immunization by discovering that light, high and low temperatures and exposure could so reduce the virulence of a microörganism that, while its injection into an animal was practically without danger or ill effect, it could still stimulate the protective mechanism of the host and induce a high degree of immunity, the two greatest advances of immunology were established. (Pasteur's discovery in 1879 was an accidental one while working with the organism of chicken-cholera.)

Vaccines may be classified according to the methods used in preparing them. Bacterins are suspensions of dead bacteria in either salt solution or broth culture. Glycerin vaccines are suspensions of dead bacteria in glycerin. Sensitized vaccines consist of dead bacteria in suspension which have been previously sensitized by antisera. Detoxicated vaccines are extracts of bacteria treated to eliminate the toxic part. Autolysates are filtrates from autolyzed bacteria. Autolyzed vaccines or artificial aggressins are suspensions of autolyzed bacteria or liquid culture media, or both. From these definitions

of the several vaccines, it will be noted that they contain bacteria, their culture media, or their autolysates.

Ectoantigens, on the other hand, are washings of bacteria prepared in such a manner that they contain the derivatives mainly of the *ectoplasm* of the bacterial cells in salt solution. From bacterins they differ by containing *no* bacteria; from glycerin vaccines, in that they contain *neither bacteria nor glycerin*; from sensitized vaccines, in that they contain *no* dead bacteria treated in any way; from detoxicated vaccines, in that the extract has been treated with nothing; from autolysates in that the method of their manufacture precludes autolysis; from autolyzed vaccines or artificial aggressins, in that they contain no autolyzed bacteria or culture media. In fact, from the foregoing it will be observed that the chief difference between ectoantigens and vaccines, so far as the physical characteristics of these are concerned, is that the former contain *only the substances obtained from the surface* of the unautolyzed bacteria, while the latter invariably contain the bacteria themselves, their "detoxicated" extracts, their autolysates, or their culture media.

Bacterial Vaccines.—The term bacterial vaccine has now become widely used to designate bacterial suspension prepared for purposes of immunization. Bacterial vaccine therapy differs from serum therapy in that the vaccine therapy is the more active form of immunization, while serum therapy is a process of passive immunization. For the purposes of this work two forms of bacterial vaccine administration are required—those which are introduced under the skin, in which suspensions of killed bacterial bodies or their ectoantigens are employed, and those which are introduced in the rectum, in which viable forms are employed.

In the first instance the doses are usually given in multiples of millions, while in the second instance the doses are usually given in multiples of trillions. In the administration of viable organisms by rectum, while the term "vaccine" would be proper where the organism used is for the purpose of establishing an immunity to overcome an infection, the term is sometimes employed with those bacterial instillations that are given for antagonistic action, used mainly for the purpose of

breaking up symbiotic cliques of toxic natures. In the latter instance, however, I prefer to use the term "antagonistic bacterial instillation," reserving the term "bacterial vaccine" for those of straight vaccines which are introduced under the skin or by rectum.

Methods of Administration—*Subcutaneous Route.*—

It has been suggested elsewhere in this volume that more or less immunity to infecting organisms in the intestinal canal may be accomplished by their administration by the subcutaneous route, in which the antibodies made in the body are discharged into the digestive canal with the abundance of secretion necessary for digestion.

Without going into all of the data connected with the manufacturing of vaccines, the method I employ is to isolate the straight strain, accomplish the most abundant growth possible, and undertake to obtain a concentrated suspension. This is manipulated in the usual way of breaking up the bacterial clumps, the organisms being killed by heat, and the vaccine tested on subsequent media to prove their non-viability. The reason of this concentrated suspension is because most of the intestinal organisms are of low virulence, and in the step-up to larger doses, commonly quantities as high as 1, 2, or 3 c.c. are necessary for the one injection.

The procedure followed is to pay no attention to the numbers of bacterial bodies in a given unit of the fluid but to accept it as it is made and to begin the injections with 1 minim, this being doubled at each injection, usually at intervals of twice a week, and the dose controlled according to the local reaction of redness, nodules, pain, etc., and the general reaction of malaise, etc. It must be evident that there is considerable variation in the pathogenic activity of different microorganisms, and to be practical in bacterial work, the method suggested serves to best clinical purpose for the subcutaneous administration. Not uncommonly, of course, various types of mixed vaccines are employed, there seldom being more than three generic types used at the one time, because of the diminished amount of agglutinin results when many strains are given at the same time.

In all of this work autogenous vaccines are employed. However, it sometimes happens that a stock form would be more active, in which case the stock employed is the same organism always from an intestinal source.

There is no doubt that the injection of dead bacteria in vaccines under the skin may and commonly does produce specific antibodies. The mechanism is similar to that taking place during infection with living microorganisms and involves the first principle of immunity. There is no doubt, however, that the pathogenic powers of a vaccine given subcutaneously are often distinctly inferior to the living antigen administered by the rectal route. It is for this reason that the rectal route is often preferred to the subcutaneous, although at times the subcutaneous serves to good purpose, especially as one part in the multiple biologic handling of a case. Another reason is that, as curative agents administered under the skin, vaccines are mostly efficacious in those infections in which phagocytosis is known to be chiefly concerned in the defense of the host. Since the mucous membrane of the intestinal canal is no doubt the most active source for the manufacturing of phagocytes, and since during the active process of digestion a large part of the blood, carrying the white as well as the red cells, is increased in volume of circulation in the tissues of the digestive tract, there is no better way to accomplish a phagocytic response than introduction of the bacterial substance into the intestine.

In no part of this work am I willing to believe that benefits which were accomplished were not mostly due to specifically direct and collateral immunization, or that the results accomplished were due only to non-specific activity. In every form of bacterial infection, however, a certain degree of success may be credited to vaccine therapy, due not only to specific effect, but to non-specific biological effects as well.

Failure to obtain beneficial therapeutic results is generally due to the use of bacteria not suitable for subcutaneous injection, to the non-accomplishment of immunity responses, or to the wrong bacteria for the infection having been employed.

Much has been said pro and con on the use of various bacterial vaccine methods. Most of that which has been in oppo-

sition is due to lack of knowledge on the part of the physician on the subject of biologic immunities, to the use of improper vaccines, or their administration in too small-sized doses. It has always been a cause for wonderment to me to see the size doses and the types of organisms recommended and used to accomplish beneficial effects in intestinal toxemia. Not uncommonly patients have claimed benefit from these, but these benefits have come mostly, when claimed, by God's grace of luck upon ignorance, or mental effect.

In the use of vaccines by different medical men no one could help but be impressed by the almost total absence of knowledge of biology, matters pertaining to infection immunities, and biologic therapy. About all that many of those who employ vaccines know of these subjects is the reading matter on the printed slips found in the containers holding stock vaccines as supplied by the vaccine manufacturing concern.

What is read is then taken for granted and then if the results are not forthcoming it is deducted that "vaccine therapy is of no value." This promptly presupposes that the statements on the slips are as correct as such slips should be, which at least is always far short of essential knowledge for proper conducting of biologic work. The physician, however, has to take more blame as a rule than the manufacturing concern. Generally the concern supplies a material, and in the absence of knowledge does the best it can to supply the information. They are not physicians or therapists. The physician, however, often guesses at the infection and in that sense assumes the etiological factor, or if examined for, the primary etiological factor is wrongly judged, especially in the presence of a mixed infection.

When a mixed infection exists, one or another of the organisms is judged as the basis for vaccines, especially if a streptococcus or a staphylococcus is found. The vaccine selected would then be of this organism when autogenous, or some other strain when stock, assuming the streptococcus or the staphylococcus as the main factor in the mixed infection. Added to these errors in the work is the generalized rule of so and so many millions injected instead of standardizing on re-

sistances, types of organisms, types of people, etc. In more ways than these, daily observation shows it as truth that there is no field of therapy in medicine in which less knowledge for its proper conduction exists. There are many excellent books on the subject, excellent teachers of it, and since biologic therapy represents the future of medicine, it behooves medical men who attempt biological work to become more able in it than they are today.

Up to the present time, with a properly prepared and potent vaccine, there is no contraindication to its use for intestinal work. Of course no form of bacterial administration in cases of this sort should be employed where there is tuberculosis, diabetes, carcinoma, debilitating disease, advanced nephritis, etc. In the use of the viable forms by rectal administration it is my custom not to employ any bacterial instillation when there are any open paths in the mucous membrane of the rectum, sigmoid or colon, or when bleeding hemorrhoids exist.

Apart from the local reaction to subcutaneous administration, one must watch for anaphylactic reactions and instances where the constitutional reaction is too pronounced. As a rule for subcutaneous administrations the outer aspect of the thigh or gluteal region is used, alternating on the two sides. The constitutional responses brought about are due almost entirely to the toxic split products of the vaccine and distinctly less so to the proteid of the bacterial body itself, which is especially true of rectal administrations. The toxic effects from the protein itself produce some reactions, and are capable of slight resistance stimulations, but fall far short of specific or worthwhile effects. To accomplish results in subcutaneous vaccines, bacteria of exogenous character are required for specific response.

Rectal Administration.—In the rectal administration four forms of therapy are employed—straight vaccines, biochemic alteration vaccines, ectoantigens, and the antagonisms. In the first, second and fourth, from 5 to 15 trillion of active bacteria are injected suspended in solutions, usually sugar solutions, making up the bulk of from 2 to 4 ounces. After the injection the patient rests on the left side for ten minutes. Such admin-

istrations are made twice a week and are followed, in the straight vaccines and biochemic alteration vaccines, in about six hours with a white blood-cell count during the first month of administration at least, and sometimes all the way through.

The figures are tabulated against the white blood-cell count made before the first instillation of vaccine was given. When the proper bacteria are employed, and by proper I mean those that are capable of accomplishing a hematopoietic response, the blood count should show a moderate hyperleukocytosis, or a leukopenia. The hyperleukocytosis runs from a rise of 1 to 15,000 above the original figures and the leukopenia a decrease of from 500 to 3000. As a rule whenever the white blood-cells are increased in numbers the increase is represented in lymphocytes and it is not uncommon to find a lymphocytosis with a leukopenia, the latter of which represents in my experience the best type of a hematopoietic response.

A type of bacterial vaccine therapy by the rectal route which I employ is what I designate as the biochemic alteration vaccine method. In this instance a single strain of organism or combinations of such as would be indicated are employed, all of the forms used being subcultured from two to five times, and usually in two, three, four or five different types of media. The bacterium then, which may be started from an original culture made for the purpose, would have been considerably changed biochemically and perhaps in its morphology, and would largely have been robbed of its infective virulence both intrinsically in the toxin produced by the bacterial bodies and also relatively of its original selective intestinal origin. This method of biochemical alteration, originally described in my text-book on *Intestinal Diseases*¹, has served in many instances not only in direct vaccine administration, but sometimes to accomplish the establishment of attenuated types of organisms instead of those originally infecting the host, the former being less toxic.

It no doubt will be suspected that to employ some of the organisms in viable form for rectal administration, especially

¹ Bassler: *Diseases of the Intestines*, 3d Ed., p. 280.

in the doses mentioned, is liable to be or would be a dangerous procedure. This is true to some extent, but not as generally as would be supposed, throughout the definitely infecting forms. One must remember that in an infection the organisms introduced for vaccine therapy would be identical with that or those existent for a time in the bowel, and in chronic infections for a long time. In this long-time presence, the bowel locally and the body generally have acquired a greater or less tolerance, and increased amounts of the same organisms in viable form do not cause the dangerous results that would be supposed. Of course here it is most essential that the biologic diagnosis be absolutely correct, especially in the highly infective organisms, because to introduce a highly infective organism when the same had not been present in the bowel for a long time or is not present at the time, would be a most dangerous procedure.

While at times in some cases large-sized doses of such organisms were employed initially, most often I have started with smaller doses and worked to the larger quantities, although lately with the highly infective types, the ectoantigens, bio-chemic alteration vaccines, subcutaneous vaccines or the antagonisms are being more often used. Not infrequently, however, only the rectal vaccines will give most satisfactory results, and in these there are two rules of safety in procedure: A, to use a minimum quantity as mentioned above, perhaps beginning with a few hundred million and rapidly increasing; and B, to pass the growths through several generations (sometimes in the same, sometimes in different, media) to rob the organisms of their specificities insofar as the intestine is concerned (B. A. V.).

Necessity of Fresh Vaccine.—It is absolutely essential in all of vaccine therapeutic work that a parent strain of vaccine should not be employed for too long a time. After years of experience with vaccines in the treatment of intestinal toxemias, new isolations and vaccines must be made for subcutaneous administration once every thirty days, and for the rectal route once every other time, and in at least half of the instances, a new vaccine is made for every single rectal instillation. This latter entails a tremendous amount of laboratory work, but it

represents the most efficient method of therapy and is the one that I strongly advise.

Experience in clinical and biologic results accomplished proves that to use a parent culture which has been standing in the icebox a week or more for inoculation into media results in the growth of the bacteria being modified very considerably for results when instilled into the rectum, and hence the necessity of making isolations and fresh vaccines every few days. The routine procedure in rectal administration today when vaccines by the rectal route are employed, is to make a fresh vaccine twice a week from fresh stool specimens and to employ them until the check-up of the infecting organism shows them to be distinctly under control. This may be accomplished definitely by the eighth instillation and it may not be accomplished short of four or five months of biweekly injections.

As a rule, the blood count after the administrations of vaccines by the rectal route is not kept up longer than during the first month, namely after the eighth instillation. The reason for this is because, as immunities are established with many bacteria, the blood responses become negative to vaccine administration. Of course, it is not uncommon, when both active immunity by subcutaneous vaccine administration and rectal administration are engaged in for the purpose of breaking up the symbiotic cliques, that four injections are made in the week, there having to be an interval of at least twenty-four hours between them.

In 1901 Wright¹ described what he termed the "negative phase" (since renamed by him "apophylactic phase") following the injection of a dose of vaccine. This represents a temporary period in which there is diminished blood resistance or lowered immunity. This period of lowered resistance is believed by Wright, and myself, to last from several hours to a day or more, followed by a positive phase of increased resistance. Wright also found that this lowered resistance was most pronounced after the injection of large doses or giving vaccines at too short intervals. There is no doubt that one of the

¹ Wright: *The Lancet*, Sept. 14, 1901; *British Med. Journal*, 1, 1069, 1903.

most important matters connected with vaccine therapy is that doses of vaccines be at proper intervals and in proper amounts when the subcutaneous route is employed—the latter being not so important when the rectal route is used.

Many of the good results in my work have come about by being most particular that the vaccine injections occur at stated intervals in the week, and while I am not prepared to state this definitely when subcutaneous administrations are employed, I do definitely state that at about seventy-two hours after the administration of vaccines by rectum the end of the negative phase is reached, the latter being the proper time for the next dose of vaccine to be given.

When rectal vaccines are given at shorter intervals than this, and shorter intervals are impossible when vaccines are made each time from fresh specimens of stool, the results in accomplishing immunity are more or less thwarted.

The production of immunity by the author's rectal vaccine method, as well as the subcutaneous, may be graphically expressed as follows:

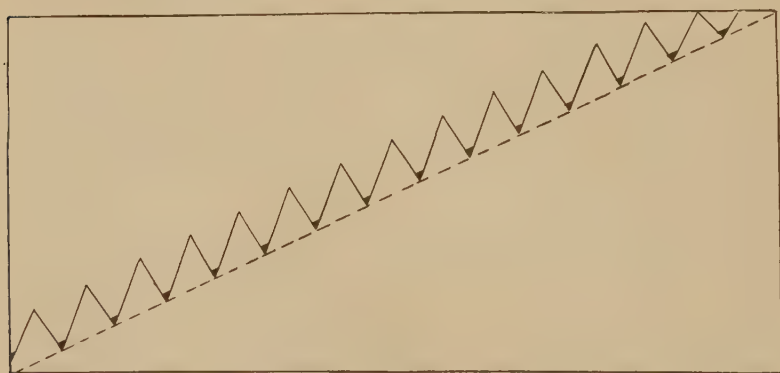


Fig. 16.—Immunity Production by Bacterial Vaccines.

▼ Vaccine injections. Ascending line, positive phase. Descending line, negative phase. Dotted line, immunity production.

Causes of Failures in the Use of Vaccines.—I desire to repeat that two men may use vaccines against an infection, one accomplishing a cure or a marked benefit, the second an absolute *nil* effect. There are many reasons for this and they are

bound up in poor work that is done in identifying the infection, the use of improper vaccines—the doses being too small, too large, or too frequently repeated—and the greatest of all factors, namely, that of not judging the individual's ability in hematopoietic response by testing him and studying him in this connection, and the lack, on the part of the administrator, of the fundamental knowledge required for immunity and biologic therapy.

The sooner the medical profession becomes a student of these types of therapy and gets away from the idea that the use of a vaccine is merely a dose of this or that, usually some stock vaccine, just as one would give a dose of this or that medicament, the sooner this whole line of work will be placed upon the high plane of therapy which it deserves.

There is no one thing in medicine more outcriing today than that biologic methods of therapy are slovenly done, largely based upon error and even more so upon ignorance and chance. With the knowledge of infection immunity and biologic therapy possible of being gained today, through numerous excellent books on the subject and from (here and there) real masters of the work, this should be corrected. To decry these methods of treatment on the basis of today's arguments, and as this work is being done, is an indictment of the knowledge of the man who makes the statement. Other than the use of insulin in diabetes (which is a biologic method of therapy) the only advances of recent years in medicine have been along the following lines of treatment.

To refresh the reader's mind I will record a few: Vaccination against rabies, against smallpox, against typhoid and paratyphoid fevers, against typhus fever, against pneumonia, against plague, against cholera, against dysentery, against common colds, against pertussis, against cerebrospinal meningitis, against scarlet fever, against acute anterior poliomyelitis, against chicken-pox, against measles, yellow fever, anthrax, etc. Added to these is the use of sera both in the prophylaxis and treatment of disease such as diphtheria, meningitis, bacillary dysentery, gas gangrene, etc., and lastly in the scientific handling, both in diagnosis and treatment of the chronic in-

testinal toxemias which are fundamentally infective processes. In this last mentioned the biologic work represents the only sane, worth-while and substantially curative method of handling the disorder, difficult, trying, time-consuming and expensive though this may be.

BACTERIAL VACCINES AND BIOCHEMIC ALTERATION VACCINES EMPLOYED IN TREATMENTS.

The vaccines I have employed rectally and subcutaneously, and would use if called for, are the following. They are represented in numbers for convenience in recording, to which *R* for rectal and *S* for subcutaneous can be added as the case may be. Those that are employed rectally, but not until they had been grown on several different media to diminish the specificity and toxicity, are marked B. A. V. (biochemic alteration vaccine).

Vaccine No. 1	<i>Achromobacter metadiffuens.</i> B. A. V.
" No. 2	<i>Achromobacter paradysentericus.</i>
" No. 3	<i>Aërobacter aërogenes.</i> B. A. V.
" No. 4	<i>Aërobacter diffuens.</i> B. A. V.
" No. 5	<i>Alcaligenes fæcaloides.</i> B. A. V.
" No. 6	<i>Alcaligenes fæcalis.</i>
" No. 7	<i>Alcaligenes gintottensis.</i> B. A. V.
" No. 8	<i>Bacillus cavacidus.</i>
" No. 9	<i>Bacillus centrosporus.</i>
" No. 10	<i>Bacillus furcosus.</i>
" No. 11	<i>Bacillus mesentericus.</i> B. A. V.
" No. 12	<i>Bacteroides acuminatus.</i>
" No. 13	<i>Bacteroides bullosus.</i> B. A. V.
" No. 14	<i>Bacteroides cornutus.</i>
" No. 15	<i>Bacteroides dimorphus.</i> B. A. V.
" No. 16	<i>Bacteroides pseudoramosus.</i>
" No. 17	<i>Bacteroides thetaiotaomicron.</i> B. A. V.
" No. 18	<i>Bacteroides variabilis.</i> B. A. V.
" No. 19	<i>Bacteroides variegatus.</i> B. A. V.
" No. 20	<i>Clostridium dissolvens.</i>
" No. 21	<i>Diplobacillus pneumoniae.</i>
" No. 22	<i>Eberthella alkalescens.</i>
" No. 23	<i>Eberthella belfastiensis.</i>
" No. 24	<i>Eberthella bentotensis.</i> B. A. V.
" No. 25	<i>Eberthella chylogena.</i>
" No. 26	<i>Eberthella dispar.</i>
" No. 27	<i>Eberthella douglasi.</i>
" No. 28	<i>Eberthella dubia.</i> B. A. V.

- Vaccine No. 29 *Eberthella dysenteriae* (Shiga-Kruse and Y of Hiss and Russell). B. A. V.
- “ No. 30 *Eberthella enterica*. B. A. V.
- “ No. 31 *Eberthella kandiensis*. B. A. V.
- “ No. 32 *Eberthella lunavensis*. B. A. V.
- “ No. 33 *Eberthella metadysenteriae*, Var. A. B. A. V.
- “ No. 34 *Eberthella metadysenteriae*, Var. B. B. A. V.
- “ No. 35 *Eberthella metadysenteriae*, Var. C. B. A. V.
- “ No. 36 *Eberthella metadysenteriae*, Var. D. B. A. V.
- “ No. 37 *Eberthella oxygenes*. B. A. V.
- “ No. 38 *Eberthella oxyphila*.
- “ No. 39 *Eberthella paradysenteriae*. B. A. V.
- “ No. 40 *Eberthella pritznitzii*.
- “ No. 41 *Eberthella pyogenes*. B. A. V.
- “ No. 42 *Eberthella talavensis*. B. A. V.
- “ No. 43 *Eberthella tangallensis*. B. A. V.
- “ No. 44 *Eberthella typhi*.
- “ No. 45 *Erythrobacillus prodigiosus*. B. A. V.
- “ No. 46 *Escherichia acidi-lacti*.
- “ No. 47 *Escherichia carolinus*.
- “ No. 48 *Escherichia coagulans*. B. A. V.
- “ No. 49 *Escherichia coli*. B. A. V.
- “ No. 50 *Escherichia colitropicalis*. B. A. V.
- “ No. 51 *Escherichia coloides*, Variety A. B. A. V.
- “ No. 52 *Escherichia coloides*, Variety B. B. A. V.
- “ No. 53 *Escherichia communior*.
- “ No. 54 *Escherichia galactophila*.
- “ No. 55 *Escherichia gastrica*. B. A. V.
- “ No. 56 *Escherichia iliaca*. B. A. V.
- “ No. 57 *Escherichia metacoli*. B. A. V.
- “ No. 58 *Escherichia metacoloides*. B. A. V.
- “ No. 59 *Escherichia neapolitana*. B. A. V.
- “ No. 60 *Escherichia paracoagulans*.
- “ No. 61 *Escherichia paraentericus*. B. A. V.
- “ No. 62 *Escherichia paragrünthali*. B. A. V.
- “ No. 63 *Escherichia pseudocoli*. B. A. V.
- “ No. 64 *Escherichia pseudocoliformis*. B. A. V.
- “ No. 65 *Escherichia pseudocoloides*, Var. A. B. A. V.
- “ No. 66 *Escherichia pseudocoloides*, Var. B. B. A. V.
- “ No. 67 *Escherichia pseudocolumbensis*.
- “ No. 68 *Escherichia pseudodysenteriae*. B. A. V.
- “ No. 69 *Escherichia schaefferi*. B. A. V.
- “ No. 70 *Escherichia vesiculosus*. B. A. V.
- “ No. 71 *Escherichia werahensis*. B. A. V.
- “ No. 72 *Escherichia wesenbergi*. B. A. V.
- “ No. 73 *Escherichia wesenbergoides*. B. A. V.
- “ No. 74 *Micrococcus ovalis*.
- “ No. 75 *Proteus asiaticus*. B. A. V.
- “ No. 76 *Proteus vulgaris*.

Vaccine	No. 77	<i>Pseudomonas</i> <i>aëruginosa</i> .	B. A. V.
"	No. 78	<i>Salmonella</i> <i>guimai</i> .	
"	No. 79	<i>Salmonella</i> <i>mobilis</i> .	B. A. V.
"	No. 80	<i>Salmonella</i> <i>negombensis</i> .	B. A. V.
"	No. 81	<i>Salmonella</i> <i>para-aërtrycke</i> .	B. A. V.
"	No. 82	<i>Salmonella</i> <i>para-asiatica</i> .	B. A. V.
"	No. 83	<i>Salmonella</i> <i>paradiffuens</i> .	B. A. V.
"	No. 84	<i>Salmonella</i> <i>pseudo-asiatica</i> .	B. A. V.
"	No. 85	<i>Salmonella</i> <i>pseudomorgani</i> .	B. A. V.
"	No. 86	<i>Salmonella</i> <i>pseudowesenbergi</i> .	B. A. V.
"	No. 87	<i>Salmonella</i> <i>Schotmülleri</i> A.	B. A. V.
"	No. 88	<i>Salmonella</i> <i>Schotmülleri</i> B.	B. A. V.
"	No. 89	<i>Salmonella</i> <i>veboda</i> .	B. A. V.
"	No. 90	<i>Salmonella</i> <i>watareka</i> .	B. A. V.
"	No. 91	<i>Salmonella</i> <i>willegodai</i> .	B. A. V.
"	No. 92	<i>Serratia</i> <i>haveniensis</i> .	
"	No. 93	<i>Staphylococcus</i> <i>albus</i> .	B. A. V.
"	No. 94	<i>Streptococcus</i> <i>anginosus</i> .	B. A. V.
"	No. 95	<i>Streptococcus</i> <i>equi</i> .	B. A. V.
"	No. 96	<i>Streptococcus</i> <i>equinus</i> .	B. A. V.
"	No. 97	<i>Streptococcus</i> <i>faecalis</i> .	B. A. V.
"	No. 98	<i>Streptococcus</i> <i>hemolyticus</i> .	B. A. V.
"	No. 99	<i>Streptococcus</i> <i>hemolyticus</i> II.	B. A. V.
"	No. 100	<i>Streptococcus</i> <i>hemolyticus</i> III.	B. A. V.
"	No. 101	<i>Streptococcus</i> <i>ignavus</i> .	B. A. V.
"	No. 102	<i>Streptococcus</i> <i>infrequens</i> .	B. A. V.
"	No. 103	<i>Streptococcus</i> <i>mitis</i> .	B. A. V.
"	No. 104	<i>Streptococcus</i> <i>non-hemolyticus</i> I.	B. A. V.
"	No. 105	<i>Streptococcus</i> <i>non-hemolyticus</i> II.	B. A. V.
"	No. 106	<i>Streptococcus</i> <i>non-hemolyticus</i> III.	B. A. V.
"	No. 107	<i>Streptococcus</i> <i>pyogenes</i> .	B. A. V.
"	No. 108	<i>Streptococcus</i> <i>salivarius</i> .	B. A. V.
"	No. 109	<i>Streptococcus</i> <i>subacidus</i> .	B. A. V.

NOTE: See description of organisms in Chapter X for further biological matters, also Ectoantigens, Antagonisms and Sera in this chapter.

BACTERIAL ECTOANTIGENS EMPLOYED IN TREATMENT.

Ferry and Fisher¹ made comparative studies of the immunizing values of various antigens prepared from toxin-producing bacteria. They found that twenty-four hour broth centrifugates contain antigens which will stimulate the formation of immune bodies of much higher titer than those stimulated by

¹ Ferry and Fisher: British Jour. of Exper. Path., 1924; vol. v, pp. 185, 205, Apr. 12, 1924.

centrifugates of cultures five or ten days old, or by antigens composed of sedimented bacteria. It appeared as though such antigens were not entirely endotoxic in nature, nor were they typical autolysates or aggressins. These antigens, or antigens prepared in this way, are associated with the extra-cellular structure of organisms, the ectoplasm.

In order to obtain this antigen from the bacterial cells the organisms are grown in the usual ways on solid media for twenty-four hours, washed off with physiologic salt solution, at once agitated for fifteen minutes in a mechanical shaker and immediately run through a Sharples centrifuge, the whole operation taking about thirty minutes. This rapid handling precludes the disintegration of the organisms to any practical extent, thus preventing the liberation of the endotoxin and also the formation of autolysates; 0.5 phenol is added as a preservative. Such ectoantigens have been proven by these investigations to be of value in the washings with the pneumococcus Type I, paratyphoid A, type strains of *Escherichia coli*, streptococcus, gonococcus and staphylococcus.

The more simplified method of securing soluble specific substances from various bacteria, recently advanced by Ando¹ has also been used in this connection. It does away with the necessity of agitation and filtration, and in my experience so far is satisfactory for this work. Since, in all specific substances from bacteria when arranged for in treatment, standardization is more or less guess work, it is essential to begin administrations, whether introduced by rectum or under the skin, in guarded ways. In calculating this, the bacteria or the types being used are important in the preliminary estimations. The most important, however, are the reaction experiences with the individual patient.

This form of treatment has been added to the work done these last few years. Rarely has it been depended upon alone. As time goes on and experience accumulates it is serving to more and more purpose in handling infections of aërobic bacteria in the intestinal canal, especially those of the exotoxic

¹ K. Ando: Jour. of Immunology, vol. xvii, No. 6, Dec., 1929.

class, by the subcutaneous method of administration. Not infrequently the ectoantigens have been used with the more strictly endotoxic organisms. While this appears quite academic, so many clinically worth-while results have been accomplished that either what were believed to be endotoxic organisms are not so, or at least so only in part (and therefore have secreted toxic factors), or some general hematopoietic response accomplished the benefits.

The use of the ectoantigens, as well as the subcutaneous vaccines, raises a most interesting question, one quite impossible of proving so far as the infections locked up in the general body are concerned, and most especially so with the intestines. How can an immunizing substance, whatever its nature may be, introduced under the skin, into the muscles or even the veins, bring about a change in the biology of the intestinal canal? One's first thought is that the contents of the intestinal canal are not an integral part of the body and not a body tissue. Therefore they do not come directly in contact with substances that are in the blood or lymph, for comparatively these intestinal contents are quite outside the body while still in it. That these changes have been so frequently accomplished proves conclusively that immune substances are also contained in the fluids of the gastric juice perhaps, but more particularly in the bile, pancreatic juice, the succus entericus, and the intestinal fluids in general, which totally make a steady secretion all the way down the tract.

So prompt and striking have some of the changed biologic results been in the intestinal contents on one or two injections that I am now distinctly of the belief that these digestive secretions not only serve the purposes of digestion but also are avenues of elimination of many soluble products of the blood of no small moment. With the substances thus eliminated are the immune factors (both those introduced and those responding from them in the body) in large part, and they, mixing with the secretions, exert an inhibitory influence on the bacteria present in the bowel at the time. Certainly the urine, the sweat and the exhaled air do not contain these substances,

as experiments will prove, although a slight amount does come out in the urine.

In my belief they are eliminated by the intestinal secretions in the largest amounts and therefore treatment work along these lines when indicated, wholly or in part, is most valuable.

The ectoantigens so far used and would be used if called for, with their symbols, are the following. As with the vaccines, *R* may be added for rectal, and *S* for subcutaneous.

Ectoantigen	No. 1	<i>Achromobacter paradysentericus</i> .
"	No. 2	<i>Aërobacter aërogenes</i> .
"	No. 3	<i>Aërobacter diffuens</i> .
"	No. 4	<i>Alcaligenes fæcaloides</i> .
"	No. 5	<i>Alcaligenes fæcalis</i> .
"	No. 6	<i>Alcaligenes gintottensis</i> .
"	No. 7	<i>Bacillus cavidus</i> .
"	No. 8	<i>Bacillus mesentericus</i> .
"	No. 9	<i>Bacteroides acuminatus</i> .
"	No. 10	<i>Bacteroides bullosus</i> .
"	No. 11	<i>Bacteroides variegatus</i> .
"	No. 12	<i>Diplococcus pneumoniae</i> .
"	No. 13	<i>Eberthella alkalescens</i> .
"	No. 14	<i>Eberthella bentotensis</i> .
"	No. 15	<i>Eberthella chylogena</i> .
"	No. 16	<i>Eberthella dispar</i> .
"	No. 17	<i>Eberthella dubia</i> .
"	No. 18	<i>Eberthella enterica</i> .
"	No. 19	<i>Eberthella kandiensis</i> .
"	No. 20	<i>Eberthella metadysenteriae</i> , Variety A, B, C and D.
"	No. 21	<i>Eberthella oxyphila</i> .
"	No. 22	<i>Eberthella paradysenteriae</i> .
"	No. 23	<i>Eberthella pritznitzi</i> .
"	No. 24	<i>Eberthella pyogenes</i> .
"	No. 25	<i>Eberthella talavensis</i> .
"	No. 26	<i>Eberthella tangallensis</i> .
"	No. 27	<i>Eberthella typhi</i> .
"	No. 28	<i>Erythrobacillus prodigiosus</i> .
"	No. 29	<i>Escherichia acidi-lacti</i> .
"	No. 30	<i>Escherichia carolinus</i> .
"	No. 31	<i>Escherichia coagulans</i> .
"	No. 32	<i>Escherichia coli</i> .
"	No. 33	<i>Escherichia colitropicalis</i> .
"	No. 34	<i>Escherichia coloides</i> , Variety A and B.
"	No. 35	<i>Escherichia communior</i> .
"	No. 36	<i>Escherichia galactophila</i> .
"	No. 37	<i>Escherichia gastrica</i> .

Ectoantigen	No. 38	<i>Escherichia iliaca</i> .
"	No. 39	<i>Escherichia metacoli</i> .
"	No. 40	<i>Escherichia metacoloïdes</i> .
"	No. 41	<i>Escherichia paraentericus</i> .
"	No. 42	<i>Escherichia paragrünthali</i> .
"	No. 43	<i>Escherichia pseudocoli</i> .
"	No. 44	<i>Escherichia pseudocoliformis</i> .
"	No. 45	<i>Escherichia pseudocoloides</i> , Var. A and B.
"	No. 46	<i>Escherichia pseudocolumbensis</i> .
"	No. 47	<i>Escherichia schaefferi</i> .
"	No. 48	<i>Escherichia vesiculosus</i> .
"	No. 49	<i>Escherichia wesenbergi</i> .
"	No. 50	<i>Escherichia wesenbergoides</i> .
"	No. 51	<i>Pseudomonas aëruginosa</i> .
"	No. 52	<i>Salmonella guimai</i> .
"	No. 53	<i>Salmonella negombensis</i> .
"	No. 54	<i>Salmonella pseudomorgani</i> .
"	No. 55	<i>Salmonella pseudowesenbergi</i> .
"	No. 56	<i>Salmonella Schotmülleri</i> , A and B.
"	No. 57	<i>Salmonella veboda</i> .
"	No. 58	<i>Salmonella watareka</i> .
"	No. 59	<i>Salmonella willegoidai</i> .
"	No. 60	<i>Staphylococcus</i> , all forms.
"	No. 61	<i>Streptococcus anginosus</i> .
"	No. 62	<i>Streptococcus equi</i> .
"	No. 63	<i>Streptococcus equinus</i> .
"	No. 64	<i>Streptococcus fæcalis</i> .
"	No. 65	<i>Streptococcus hemolyticus</i> .
"	No. 66	<i>Streptococcus hemolyticus</i> II.
"	No. 67	<i>Streptococcus hemolyticus</i> III.
"	No. 68	<i>Streptococcus ignavus</i> .
"	No. 69	<i>Streptococcus infrequens</i> .
"	No. 70	<i>Streptococcus mitis</i> .
"	No. 71	<i>Streptococcus non-hemolyticus</i> I.
"	No. 72	<i>Streptococcus non-hemolyticus</i> II.
"	No. 73	<i>Streptococcus non-hemolyticus</i> III.
"	No. 74	<i>Streptococcus pyogenes</i> .
"	No. 75	<i>Streptococcus salivarius</i> .
"	No. 76	<i>Streptococcus subacidus</i> .

NOTE: See description of organisms in Chapter X for further biological matters, also Vaccines, Antagonisms, and Sera in this chapter.

Recently ectoantigens have also been introduced by rectum. They have been prepared autogenously in bulk, iceboxed and given in doses ten to twenty times that of the subcutaneous method, made up to 2 to 4 ounces quantity by normal saline solution as diluent for injection. Here and there an ecto-

antigen from a stock strain may be employed for a short run. In breaking up inimical biological cliques short runs of the less important organisms in the associations are often called for, these types of treatment usually being more or less of secondary importance as compared to the organisms of the infection, be they single or mixed. This same rule is often called for with the ectoantigen by subcutaneous route. In the major infections the vaccines are generally used instead of ectoantigens.

As experience multiplies, however, the subcutaneous use of ectoantigens often serves to better purpose than the vaccines in organisms of the *Eberthella*, *Escherichia*, *Salmonella*, *Streptococcus*, *Staphylococcus*, *Achromobacter*, and *Alcaligenes* groups. No set rule on the initial choice between vaccines or ectoantigens can be expressed, nor is there any set rule or preference between their administrations subcutaneously or rectally. One of the main factors of difficulty here is that in both vaccines and ectoantigens when given subcutaneously, the doses (initially at least) are much smaller than by the rectal route.

The biologic diagnosis having been made and the organisms in incidence, importance and associations listed, one plots the biologic therapeutic treatments according to the infections found. Since all sorts of infections and combinations are met with, so different combinations of biologic handling are planned. It is not uncommon in one case to use the ectoantigen of one organism and the vaccine of another subcutaneously, and also use ectoantigen, vaccine, biochemic altered vaccine, organisms for antagonistic action, with perhaps sera in addition, used more or less or at different times in the treatment.

Obviously, when possible, it is best to keep the plan relatively simple and not complicate the therapy by too many biological treatments going on at the same time. It is occasionally seen that the control of one organism by a single method will cause the breakdown of a perhaps difficult to figure and understand multiple associated infection. This, however, does occur often, either because too much attention was paid to some organism other than to the more important one, or because the biologic method of approach and character is not efficient, or the selection is not as efficient as one of different approach and

character would be. Thus after the original biologic therapeutic set-up has been conducted for a while, check-up of the biological picture is called for. This is done at latest during the fourth week of treatment, and it is at this time that original mistakes can be corrected, changes in the bacteriology can be noted, and biologic procedures changed, added to, or continued.

Such check-ups are called for after even biologically satisfactory results, and irrespective of the patient's satisfaction with clinical results. It is not unusual in the first month to have a complete biological check-up each week. It is not uncommon for a dozen different organism selections and procedures to have been followed before termination of treatment. The average in my last one thousand straight cases was slightly over seven different procedures from the beginning to the end of treatment.

When the ectoantigens are employed in rectal instillations, estimation of dosages is almost impossible. One has to figure in a relative way on the abundance of the surface growth and the amounts of surface growth employed. Obviously the amounts introduced rectally would be many times that employed subcutaneously, and to accomplish this, extensive areas of surface growths are required. Since this means a large amount of work and time, subculturizations from parent cultures are often necessary, these being used for a length of time before re-isolation.

BACTERIAL ASSOCIATIONS. ANTAGONISMS. THOSE EMPLOYED IN TREATMENT.

One microbe may favor the growth and activity of another, or both may be benefited by the combination, the latter process being generally spoken of as symbiosis. The terms antagonism or antibiosis are employed for the occurrences where there is a clearly demonstrable harmful effect of one microörganism on another or when a characteristic product fails to be formed or disappears in the mixed culture. The antagonism between different forms and strains of intestinal bacteria has been taken advantage of for the attainment of clinical results. Only the

portions of this biologic work pertaining to the intestinal canal will be presented in this volume.

Of first significance here is that the *Erythrobacillus prodigi-
osus*, when added to the *Clostridium œdematis maligni* and
growing in association with it, invariably produces a marked
type of intestinal toxemia. The same is true when the *Pro-
teus vulgaris* exists with the above-named *Clostridium*. This
is also true with the *Proteus vulgaris* and most of the aërobes
of the intestinal canal. Rhein used the *Alcaligenes fæcalis* in
bouillon to encourage anaërobic growth, and found when this
organism is numerous in the gut, excessive anaërobic activity
with toxicity is generally present.

When the *Clostridium welchii* and the *Alcaligenes fæcalis*
are present in activity in the bowel the *Clostridium butyricum*
enhances their growth so that they will grow under anaërobic
conditions, and therefore are doubly toxic. The *Proteus vul-
garis* increases the virulence of the *Clostridium welchii*, the
Clostridium œdematis maligni and others. There is also an
enhancing situation between the *Clostridium putrificus* and
certain anaërobic organisms of the intestinal canal.

The above action has been used therapeutically in certain
organisms of the aciduric group. Starting from the work of
Metchnikoff with *B. bulgaricus*, it was soon found that this
organism could not be implanted in the intestinal tract and
Lactobacillus acidophilus, a normal inhabitant, was substituted
on the belief that the *Bacteroides bifidus*, found in the intes-
tines of breast-fed infants, is responsible for inhibition of the
growth of other bacteria, and its acid products were thought
to be mild stimulants to the bowel walls. Thus the implanta-
tion of the *Bacillus acidophilus* was suggested as a thera-
peutic measure. This was done by Rettger and Cheplin in
1921, fourteen years after C. Herter distinctly drew attention
to it.

According to Rettger and Cheplin the antagonistic action
of the *Bacillus acidophilus* has been abundantly proved against
putrefactive anaërobes, *Escherichia coli* and many other bac-
teria. While there is more or less laboratory substantiation
of this, practically it is a clinical myth. In the first place

the organisms of the *Escherichia coli* group, with some exceptions here and there, are fundamentally not proteolytic in action. The *Escherichia coli* require the association of certain of the anaërobes for the production of toxic bodies which the anaërobes prepare initially and the *Escherichia coli* only finally split the proteins off. Therefore, the *Escherichia coli* are rather innocent in the production of the condition, the anaërobes being by far the more important class, and against these strict anaërobes of the intestinal canal the *Bacillus acidophilus* has no antagonistic value.

There seemingly is an antagonistic action between the *B. mesentericus* and various types of *Streptococci*, the latter of which have been taken advantage of in my work with apparent success in individual instances. There is an antagonistic action between certain strains of the *Staphylococcus albus*, occasionally found in the bowel, and numerous strains of well-identified *Pneumococci*, not an uncommon denizen in the intestinal canal and one of significance in toxic ways.

There are a number of antagonisms between various organisms of the *Escherichia* groupings, and when these organisms are believed to be bound up in toxemia production, laboratory studies on the feces of the individual will lead to the suggestions of strains that might be beneficial. Prell (1917) and Nissle (1916), following my work¹ of six years before, suggested the use of active cultures of *Escherichia coli* strains for inhibitory effect in the intestinal canal.

In chronic instances of the *Salmonella Schotmülleri* A and B infections, various strains of the *Escherichia coli* as shown by Theobald Smith and D. E. Smith have been used with therapeutic success. There is also a distinct antagonism, which is practically in opposite directions, between the *Clostridium welchii* and the *Bacteroides bifidus*, the *Escherichia coli* and most of the strict anaërobes, the *Bacteroides bifidus* and, to a very slight extent, the *Bacillus acidophilus*, and some of the strains of the *Escherichia coli* and *Eberthella typhi*, *Eberthella dysenteriae* and *paradysenteriae*, some *Staphylococci*, *Erythro-*

¹ Bassler: Medical Record, Sept. 24, 1910.

bacillus prodigiosus, *Pseudomonas aeruginosa* and the *Micrococcus ovalis*. What is interesting here is that some of the strains of the *Escherichia coli* require the *Bacteroides bifidus* for this inhibitory activity.

The antagonisms taken advantage of for treatment by rectal instillations have been:

<i>Infections.</i>	<i>Antagonisms.</i>
<i>Aërobacter aërogenes</i>	{ <i>Escherichia coli</i> strains 5, 6, 7, 8, 9, 16, 17, 18, 19, 20. <i>Bacteroides bifidus</i> , with or without the above.
<i>Aërobacter cloacæ</i>	{ <i>Escherichia coli</i> strains 5, 6, 7, 8, 9. <i>Bacteroides bifidus</i> , with or without the above.
<i>Alcaligenes bookeri</i>	{ <i>Escherichia coli</i> strains 5, 6, 7, 8, 9, 10, 11, 12, 13, 17, 18, 19, 20.
<i>Alcaligenes fæcaloides</i>	{ <i>Escherichia coli</i> strains 5, 6, 7, 8, 9, 10, 11, 12, 13, 14, 15.
<i>Alcaligenes gintottensis</i>	{ <i>Escherichia coli</i> strains 5, 6, 7, 8.
<i>Bacillus acidophil-aërogenes</i> ...	{ <i>Escherichia coli</i> strains 9, 10, 11, 12, 13, 14, 15.
<i>Bacillus acidophilus</i>	{ <i>Escherichia coli</i> strains 9, 10, 11, 12, 13, 14, 15.
<i>Bacillus fusus</i>	{ <i>Escherichia coli</i> strains 5, 6, 7, 8, 9, 10, 11, 12, 13, 14, 15.
<i>Bacillus lautis</i>	{ <i>Escherichia coli</i> strains 1, 2, 3, 4, 5. <i>Bacteroides bifidus</i> , with or without the above.
<i>Bacillus mesentericus</i>	{ <i>Escherichia coli</i> strains 5, 6, 7, 8, 9, 10, 11, 12, 13, 14, 15, 16, 17. <i>Bacillus acidophilus</i> .
<i>Bacillus tritus</i>	{ <i>Escherichia coli</i> strains 1, 2, 3, 4, 5, 6, 7, 8, 9. <i>Pseudomonas aeruginosa</i> .
<i>Bacteroides dimorphus</i>	{ <i>Escherichia coli</i> strains 5, 8, 9, 10, 11, 12, 13, 14, 15.
<i>Bacteroides tenuis</i>	{ <i>Escherichia coli</i> strains 5, 6, 7, 8, 9, 10, 11, 12, 13, 14, 15, 16, 17, 18, 19, 20.
<i>Clostridium butyricum</i>	{ <i>Escherichia coli</i> strains 5, 6, 7, 8, 9, 11, 12, 13, 14, 15. <i>Bacteroides bifidus</i> , with or without the above.

<i>Infections.</i>	<i>Antagonisms.</i>
<i>Clostridium coagulans</i>	{ <i>Escherichia coli</i> strains 5, 6, 7, 8, 9.
<i>Clostridium dissolvens</i>	{ <i>Escherichia coli</i> strains 5, 6, 7, 8, 9.
<i>Clostridium filamentosum</i>	{ <i>Escherichia coli</i> strains 1, 4, 5, 8, 9.
<i>Clostridium fissum</i>	{ <i>Escherichia coli</i> strains 1, 2, 3, 4, 5, 6, 7, 8, 9, 10, 11, 12, 13, 14, 15.
<i>Clostridium œdematis maligni</i>	{ <i>Escherichia coli</i> strains 1, 2, 3, 4, 5, 6, 7, 8, 9, 10, 11, 12, 13, 14, 15.
<i>Clostridium ovalis</i>	{ <i>Escherichia coli</i> strains 1, 2, 3, 5, 8, 9.
<i>Clostridium putrificum</i>	{ <i>Escherichia coli</i> strains 10, 11, 12, 13, 14, 15, 16, 17, 18, 19, 20.
<i>Clostridium regularis</i>	{ <i>Escherichia coli</i> strains 5, 8, 9, 10, 11, 12, 13, 14, 15.
<i>Clostridium saccharolyticum</i>	{ <i>Escherichia coli</i> strains 4, 5, 7, 9, 10, 11, 12, 13, 14, 15.
<i>Clostridium tetani</i>	{ <i>Escherichia coli</i> strains 1, 2, 3, 4, 5, 6, 7, 8, 9, 10, 11, 12, 13, 14, 15, 16, 17.
<i>Clostridium welchii</i>	{ <i>Escherichia coli</i> strains 5, 6, 7, 8, 9. <i>Proteus vulgaris</i> . <i>Bacteroides bifidus</i> .
<i>Clostridium zooglicum</i>	{ <i>Escherichia coli</i> strains 5, 6, 7, 8, 9.
<i>Diplococcus Gram-positive</i>	{ <i>Escherichia coli</i> strains 5, 6, 7, 8, 9.
<i>Eberthella bienstockii</i>	{ <i>Escherichia coli</i> strains 5, 11, 12, 13, 14, 15.
<i>Eberthella chylogena</i>	{ <i>Escherichia coli</i> strains 4, 5, 6, 7, 9, 10, 11, 12, 13, 14, 15.
<i>Eberthella dysenteriae</i>	{ <i>Escherichia coli</i> strains 5, 6, 7, 8, 9, 16, 17, 18, 19, 20.
<i>Eberthella kandiensis</i>	{ <i>Escherichia coli</i> strains 1, 2, 3, 4, 5.
<i>Eberthella talavensis</i>	{ <i>Escherichia coli</i> strains 1, 2, 3, 4, 5, 6, 7, 9, 10, 11, 12, 16, 17, 18, 19.
<i>Eberthella typhi</i>	{ <i>Escherichia coli</i> strains 6, 7, 8, 9, 10, 17, 18, 19, 20. <i>Bacteroides bifidus</i> , with or without the above.
<i>Erythrobacillus prodigiosus</i>	{ <i>Escherichia coli</i> strains 1, 2, 3, 4, 5, 6, 7, 8, 9, 16, 17, 18, 19, 20.
<i>Escherichia alkalescens</i>	{ <i>Escherichia coli</i> strains 1, 2, 3, 4, 5.
<i>Escherichia carolinus</i>	{ <i>Escherichia coli</i> strains 1, 2, 3, 4, 5.

*Infections.**Antagonisms.*

	<i>Pseudomonas</i> <i>aëruginosa</i>. <i>Acrobacter</i> <i>aërogenes</i>. <i>Escherichia coli</i> strains 10, 11, 12, 13, 14, 15, 16, 17, 18, 19, 20. <i>Aërobacter</i> <i>cloacæ</i>. <i>Bacillus</i> <i>acidophilus</i>. <i>Bacillus</i> <i>bulgaricus</i>. <i>Lactobacillus</i> <i>acidophilus</i>. <i>Clostridium</i> <i>butyricum</i>. <i>Erythrobacillus</i> <i>prodigosus</i>. <i>Micrococcus</i> <i>ovalis</i>.
<i>Escherichia coli</i> , 5, 6, 7, 8	
<i>Escherichia gastrica</i>	<i>Clostridium</i> <i>welchii</i>, subcultured strains. <i>Escherichia coli</i> strains 5, 6, 7, 8, 9, 10, 11, 12, 13, 17, 18, 19, 20.
<i>Escherichia metacoloïdes</i>	<i>Escherichia coli</i> strains 5, 6, 7, 8, 9, 10, 11, 12, 13, 14, 15, 16, 17, 18, 19, 20.
<i>Escherichia pseudocolumbensis</i> .	<i>Escherichia coli</i> strains 1, 2, 3, 4, 5, 6, 7, 8.
<i>Escherichia wesenbergi</i>	<i>Escherichia coli</i> strains 1, 2, 3, 4, 5, 6, 7, 8. <i>Bacteroides</i> <i>bifidus</i>.
<i>Bacillus</i> <i>acidophilus</i>	<i>Escherichia coli</i> strains 1, 2, 3, 4, 5, 6, 7, 8.
<i>Micrococcus</i> <i>ovalis</i>	<i>Escherichia coli</i> strains 5, 6, 7, 8.
<i>Monococcus</i> Gram-positive	<i>Escherichia coli</i> strains 5, 6, 7, 8.
<i>Pneumococcus</i>	<i>Staphylococcus</i> <i>albus</i>.
<i>Proteus</i> <i>vulgaris</i>	<i>Clostridium</i> <i>welchii</i> subcultured strains. <i>Escherichia coli</i> strains 5, 6, 7, 8.
<i>Pseudomonas</i> <i>aëruginosa</i>	- Some strains of the streptococci. - Strains of the staphylococci. <i>Escherichia coli</i> strains 5, 6, 7, 8, 10, 12, 15, 16, 17, 20. <i>Proteus</i> <i>vulgaris</i>. <i>Bacteroides</i> <i>bifidus</i>.
<i>Salmonella</i> <i>guimai</i>	<i>Escherichia coli</i> strains 1, 2, 3, 4, 5, 6, 7, 8, 9, 16, 17, 18, 19, 20.
<i>Salmonella</i> <i>para-aërtrycke</i>	<i>Escherichia coli</i> strains 5, 6, 7, 8, 9, 10, 11, 12, 13, 14, 15, 16, 17, 18, 19, 20.
<i>Salmonella</i> <i>para-asiatica</i>	<i>Escherichia coli</i> strains 5, 6, 7, 8, 9.
<i>Salmonella</i> <i>Schotmülleri</i> A, B ..	<i>Escherichia coli</i> strains 5, 6, 7, 8, 9, 10, 11, 12, 13, 14, 15, 16, 17, 18, 19, 20.
<i>Staphylococcus</i> , all forms	<i>Escherichia coli</i> strains 5, 6, 7, 8, 9, 10, 11, 12, 13, 14, 15, 16, 17, 18, 19, 20. <i>Bacteroides</i> <i>bifidus</i>, with or without the above.

<i>Infections.</i>	<i>Antagonisms.</i>
Streptococcus, all Gram-negative strains	{ Escherichia coli strains 5, 6, 7, 8, 9, 10, 11, 12, 13, 14, 15, 16, 17, 18, 19, 20.
Streptococcus, all Gram-positive strains	{ Pneumococcus. Escherichia coli strains 1, 2, 3, 4, 5, 6, 7, 8, 9, 10. Bacteroides bifidus. Bacillus mesentericus. Erythrobacillus prodigiosus.
Vibrio proteus	{ Streptococcus faecalis. Pseudomonas aeruginosa. Escherichia coli strains 5, 6, 7, 8, 9, 10, 16, 17, 18, 19, 20.

Sometimes in infections represented by organisms mentioned in the right hand column those in the left hand column have been used.

NOTE: See description of organisms in Chapter X for further biological matters, and Vaccines, Ectoantigens and Sera in this chapter.

Regarding the different strains of *Escherichia coli* used, various cases of identical infections have had different strains employed. It is not possible to state which one would be most effective, and this solution can only be worked out in the individual case by checking the stools closely. In most of the subculture work, the second to fifth subcultures have been employed, close checking of the feces also being required.

In almost all instances of intestinal toxemia more than one infection exists. This necessitates different types of instillations according to the infection make-up, these usually being given at the one dose although not infrequently at different times; this depends upon the bacteria employed and the reactions. If the case is properly one of primary intestinal toxemia, some results from antagonism work are to be expected. If they are not forthcoming (and close laboratory checking is the only proof of this being accomplished), either the biologic diagnosis is wrong, the laboratory work poor, the wrong antagonism is used, the biologic treatments are incomplete, or in complex set-up results cannot be accomplished.

The ability in diagnosis and treatment which comes from experience in this cannot be taught in words any more practically than an artist could detail in words how he paints a

picture. After about five years of most close work in the study of vaccines, ectoantigens, sera and their application to treatment, knowledge worth while is attained by the close student. Not a few otherwise capable men seem never to learn it. The best student I have had working in my laboratories had a fairly well developed ability in biologic work after five years of steady application to the work, this comprising a weekly average of about 200 carefully made stool examinations.

The numbering of the various strains of *Escherichia coli* represents the Dunham schema, the one used by me for years. Here and there some of these are now known to belong to a separate grouping of organism, but for simplicity the original procedure is more satisfactory and thus preserved.

All of the organisms instilled in rectal vaccine and antagonism work are in viable form, they essentially being the culture as they (or it) are taken from the incubator. The only additions made are those of sugar solutions (saccharose or dextrose) to make the injection up to from 60 to 120 cubic centimeters (2 to 4 ounces).

The organism content of the injection usually comprises from 5 to 20 trillion at an injection. When the culture employed is fluid this is easy to conduct. If growths on hard media are employed they are washed off with the sugar solution and made up in bulk to the above quantity for injection. In this it depends roughly upon the growth as to whether more than one culture tube is employed. With most of the organisms doses of less than 5 trillion are not of much value. The reason for this is that most of the intestinal organisms have a very low virulence and large-sized doses are required for results.

Here and there in the antagonism work the results can be depended upon in permanent and substantial ways. Often this is conditioned on the length of time the instillations are kept up and the size of the doses. In not a few instances, however, the results accomplished are modified, and again they may not be lasting, this being less true with the other types of biologic treatments. Close checking is required in this line of work,

and shifts into the more depended-upon immunity methods are often called for.

THE ANTIBACTERIAL SERA EMPLOYED IN TREATMENT.

Antibacterial sera are those containing immune bodies whose action is directed against bacteria as entities or against the products of their disintegration, or possibly against some product of their metabolism. The reader here notes that this action is in contradistinction to the action of antitoxins, in which the antibody acts by neutralizing a toxic principle elaborated by the organism, either as a product of metabolism or as a result of ferment action on some of the substances present in its environment.

The agglutinins and precipitins, some of which are probably identical, act directly on their antigen without the aid of a third factor. Opsonins or tropins, the so-called "protective" antibody, the complement fixation antibody, and the bacteriolytic antibodies, perform their specific function only in conjunction with complement.

The list of sera used, and their symbols, is as follows:

<i>Sera.</i>	<i>Infections.</i>
Sera No. 1. Anaërobic	Bacteroides dimorphus.
	Bacteroides lævis.
	Bacteroides liquefaciens.
	Bacteroides tenuis.
	Clostridium alcaligenes.
	Clostridium butyricum.
	Clostridium dissolvens.
	Clostridium filiformis.
	Clostridium œdematis maligni.
	Clostridium ovalaris.
	Clostridium putrificum.
	Clostridium saccharolyticum.
Sera No. 2. Antidysenteric polyvalent	Clostridium welchii.
	Eberthella bienstockii.
	Eberthella dysenteriae Shiga-Kruse and Y of Hiss and Russell.
	Eberthella enterica.
	Eberthella metadysenteriae A, B, C and D
	Eberthella paradysenteriae.
	Eberthella pyogenes.
	Eberthella, other forms occasionally.

<i>Sera.</i>	<i>Infections.</i>
Sera No. 3. Antistreptococcus ..	Streptococcus anginosus.
	Streptococcus equi.
	Streptococcus equinus.
	Streptococcus faecalis.
	Streptococcus hemolyticus.
	Streptococcus hemolyticus II.
	Streptococcus hemolyticus III.
	Streptococcus ignavus.
	Streptococcus infrequens.
	Streptococcus mitis.
	Streptococcus non-hemolyticus I.
	Streptococcus non-hemolyticus II.
	Streptococcus non-hemolyticus III.
	Streptococcus pyogenes.
	Streptococcus salivarius.
	Streptococcus subacidus.
Sera No. 4. Chantemesse	Eberthella typhi.
	Salmonella pseudomorgani.
	Salmonella schotmülleri A and B.
Sera No. 5. Pneumococcus ...	Diplococcus pneumoniae.
Sera No. 6. Tetanus antitoxin ..	Clostridium tetani.

NOTE: See description of organisms in Chapter X for further biological matters, Vaccines, Ectoantigens and Antagonisms in this chapter.

Antidysentery Serum.—The occasional use of antidysentery serum is suggested. This is largely antitoxic and specific as to the type dysentery organisms. The Eberthella paradysenteriae (Flexner) is ineffectual against the Eberthella dysenteriae (Shiga). The antitoxin prepared from the Shiga-Kruse type is not especially effective against the Y of Hiss and Russell. The Flexner, Harris, Hiss and Duval types are the most often found in this country in both the chronic and the acute dysenteric infections. Very largely the acute infections of the Shiga-Kruse (which are most amenable to sera treatments) are called for in the acute dysenteries of European and Asiatic countries. In this country this infection does not prevail so extensively as do the other forms. For this reason a polyvalent serum is the most practical to employ, although generally vaccines answer to better purpose.

Occasionally, however, the serum is worth while in the Flexner and Y infections and it sometimes is worthy of trial

in the other forms of Eberthella infections, especially when diarrhea exists. In children no benefit comes from its use. The polyvalent serum is given subcutaneously, intramuscularly or intravenously in a large initial dose, but better results are accomplished in my experience in one-sixth or one-fourth sized doses at biweekly or weekly intervals.

Antistaphylococcus Serum.—No experience has been had with antistaphylococcus serum for several reasons. Much work is required to standardize the sera on antilysin and bacterioproin content of it and the complement fixation tests are difficult to perform and not especially to be depended upon. Vaccines and ectoantigens here answer to better purpose.

Antistreptococcus Serum.—With antistreptococcus serum, however, not a few cases have been injected. This serum depends primarily upon the content of bacteriotropins, and less so on the antitoxins and bacteriolysins. It must be given intravenously. While it usually is given in 30 to 100 c.c. doses with normal salt solution it must be remembered that this is to control a rapidly advancing acute infection. For our purpose in chronic infections of the intestine 5 to 10 c.c. doses are given at biweekly or weekly intervals for several doses. Since there is a large difference in potency of this serum, because of the strains of streptococci from which it may be prepared, in an individual case it is often wise to change the preparation.

Anaërobic Serum.—Much attention was paid to the bacteriology of gas gangrene during the World War. It was found that the *Clostridium welchii* was not the sole cause of the condition, but that other spore-producing anaërobic organisms were commonly present. For a long time it was believed that these *Clostridia* were only endotoxic, but it is now known that an exotoxin is produced by the Welch and no doubt some of the others. For this purpose serum of mixed forms (*Clostridium welchii*, *Clostridium œdematis maligni* and *Clostridium œdematiens*) has been prepared and is the desirable one to use in the above infections and tried perhaps in persistent cases of the other *Clostridium* infections. After using this anaërobic serum both ways, I prefer now to employ it in deep

muscular injections in 5 c.c. quantities biweekly or weekly for several weeks.

The *Clostridium tetani* infections of the bowel are difficult of control. Tetanus antitoxin has been used on numerous cases. Care should be taken that the preparation is fresh. No laboratory-proven benefit has come so far as the *Clostridium tetani* content in the bowel is concerned, although clinical benefit has ensued. The doses are given intramuscularly after the 1 c.c. testing dose for anaphylaxis given subcutaneously. Subsequent to this about 2000 units are given biweekly or weekly for several weeks. The anaërobic serum has also been employed in this infection with apparently as worth-while results as the tetanus antitoxin.

In chronic *Eberthella* type infections the vaccines or ecto-antigens are of most value. Occasionally the serum of Chantemesse was employed without noticeable benefits.

On the whole, the use of serums to control chronic infections and toxemias arising therefrom has been the least satisfactory of all the biologic methods. In the acute and especially the pyrexia states, more benefits ensue from their use. The control of the above suggested infections of a chronic nature is mostly the control of the associations, and usually when these are successfully handled by the other biologic types of treatment, the continuation of the *Streptococci*, *Staphylococci*, dysentery organisms and the *Clostridia* disappear.

CHAPTER XII.

DIETETIC TREATMENT.

Much has been written on the dietetic handling of intestinal toxemia. Methods go all the way from one opinion to one just as reasonable directly opposite. Added to these are the innumerable recommendations of food substances or food-stuff additions, practically all of which are meat substitutes, or substances to stimulate bowel elimination. In all of this, the vegetarian, the fruitarian, the milkarian, the proteidarian (beefsteak and water diet of Salisbury) and others have, or are having, their day. Ideas die with their expounder, and then, breaking out subsequently in a new quarter, pass along in more or less epidemic ways again. The whole performance is like the view through a kaleidoscope, the red, blue, green and orange colors always being there but the whole picture changing as the speed of the instrument is increased. The variation is added to by those who claim to be able to accomplish a change in the bacterial flora by this or that type of diet or by this or that type of food addition.

Still, some degree of benefit may come from the use of diets. Generally, however, this is seldom beyond thirty days in duration, because practically all the inimical intestinal organisms and combinations of them are facultative, so that at the end of this time the toxemia reestablishes as before and oftentimes is rendered distinctly worse by continuation of set diets. An exception in small part to this is when an anticonstipation plan is employed by means of which regular movements are accomplished, because this has the effect of keeping low the degree of running toxemia and the body take-up of the resorption products.

I beg to be excused from quoting the literature of the past, in which so little that is worth while appears, and will undertake to present only my own views. In a word, there is no definite method of dieting for intestinal toxemia, and it may

also be added that there is no definitely indicated diet which would be helpful in all instances in even the same type of conditions.

The plan most useful in my experience is to arrange a diet according to the general classification of the intestinal toxemia of the saccharo-butyric, the indolic, and the mixed form, and then not to depend upon the diet for more than thirty days of time. The rule I follow is to plan a normal diet, keeping the quantity of total protein down to not more than 60 or 90 grams a day, roughing up the diet so as to overcome constipation in the presence of a colitis, and adding calories in the form of fats where there is distinct debility or loss of tissue. I recommend the use of a high protein diet for a short time in the saccharo-butyric type, a high carbohydrate in the indolic, and the employment of a diet which gives the minimum amount of food, allowing a general selection in instances of the mixed type.

By following this plan I have been fairly successful in dieting cases of intestinal toxemia and not having the task of making out an individual diet for each patient or selecting various foods for this or that case. After making out individual lists for years I have come to the conclusion that I have largely hoodwinked myself, and I believe those who still follow this plan do likewise. I now go upon the findings in the laboratory and x-ray examination and make up a diet according to the type of toxemia, the abdominal and general requirements of the individual, always encouraging the taking of sufficient amounts of food, except in cases exhibiting the mixed form.

Before presenting these forms of diet there are some matters of interest which should be mentioned. In all types of toxemia it is desirable to promote digestion and rapid absorption from the small intestine and reduce the number of putrefactive anaërobes in the ileum and colon. To avoid the possibility of infection being carried into the stomach, the preparation of food and ordinary cleanliness are very effective—it is probably better to use cooked food as much as possible. To the same purpose the mouth must receive scrupulous care.

Carious teeth, gingivitis, pyorrhea, infective tonsils, infected sinuses, all should receive attention. The patient should be taught to use a tooth brush intelligently. Mouth washes containing peroxide of hydrogen, camphophenique or a weak solution of camphenol are well worth general application in all of these people. Oral sepsis requires strict attention.

Fruits are not above suspicion, for on the surface of most raw fruit bacteria swarm, the *Clostridium oedematis maligni*, for instance, being commonly present on the banana peel and the *Clostridium putrificum* on grape skins. Raw milk always contains a large number of bacteria and often some of the putrefactive forms. Pasteurization of milk is of little value. Sterilization or the ordinary boiling kills the lactic acid formers but does not harm the spores of the putrefactive organisms. Cheese contains many putrefactive forms and is best avoided, particularly inasmuch as many of these patients lack the protective action of a normal amount of hydrochloric acid in the stomach.

Rapid digestion and resorption leave little pabulum for the putrefactive anaërobes when the food reaches the colon, or at least this should be as small as one can control it in a diet. Chief in importance is proper mastication, which largely determines the ability of the body to utilize food rapidly. When large masses of meat are swallowed they commonly appear in the feces.

The comminution of food outside of the body is not an adequate substitute, for the patient then loses the emotional stimulus to gastric digestion and also the digestive action of the saliva as well. If the stomach is hypersensitive or a status of irritability exists, it is advisable to give more meals and to administer flaxseed or some other demulcent before eating.

It is good to keep in mind that putrefaction in the intestines is somewhat directly proportional to the amount of proteins in the food. These are obtained from meat, fish, chicken and eggs, but also from legumes and other vegetables. The vegetable proteins, however, are safer than those of the animal forms of protein foods, due to the fact that vegetable proteins are not so accessible to the alimentary or bacterial enzymes and,

therefore, are not so readily decomposed, and also the fact that bacteria utilize the carbohydrate substances commonly in preference to protein.

There is some difference in the effect of carbohydrate foods. Diets of sugar, potatoes and legumes often give rise to organic acids in gases, whereas on the other hand rice, sago, tapioca and arrowroot give rise to comparatively little fermentation. As a rule the first of these should be forbidden as well as most kinds of fish, game, pork, veal, hashed meat and made-up dishes, particularly those that have stood in the icebox over night. Stews, rich gravies, soups, meat extracts, pastries, cakes, tea and coffee, are mostly taboo.

If the patient is a bed case it may be wise for a short time to adhere to a bland type of diet. Such a plan should be especially useful where a hyperesthetic condition of the stomach exists represented in excess secretion, motility or sensation, or an irritable status of the colon exists such as would cause a diarrhea or a marked spasticity. In this instance selections from the following plan serve a good purpose, it being necessary for the dietitian to keep the total food intake for the day up to the required caloric value, which in amount should be specified by the physician.

Beef juice and tea.	Two soft eggs, 120.
Beef broth with grains or egg, 75.	Golden rod eggs, 170.
Mutton broth with grains or egg, 80.	Egg nests, 142.
Chicken broth with grains or egg, 80.	Bread omelet, 164.
Veal broth with grains or egg, 80.	Egg poached in milk, 470.
Calves' foot jelly, 65.	Coddled or scrambled eggs, 180.
Chicken jelly, 65.	
Beef jelly, 65.	Raw oysters.
Cream of asparagus soup, 313.	Creamed oysters, 284.
Cream of corn soup, 250.	Oyster soup, 242.
Cream of celery soup, 160.	Oyster stew, 205.
Cream of pea soup, 215.	Clam soup, 278.
Cream of pea flour soup, 202.	Clam stew, 250.
Cream of onion soup, 300.	Clam bouillon bisque, 355.
Cream of potato soup, 220.	
Cream of rice soup, 302.	Carrots, one small one, 20.
Tomato soup with broth, 110.	Spinach chopped, 27.
Mock bisque soup, 324.	Stewed tomatoes, canned, 51.
Dried fruit soup, 209.	
Victoria soup with broth, 315.	Corn meal mush, 182.
Rice barley and tapioca jelly, 200.	Hominy mush, 200.
	Cocoa junket, 280.

- Boiled rice, 100.
 Steamed rice, 265.
 Boiled macaroni, white sauce, 250.
 Boiled macaroni, tomato sauce, 190.
 Baked macaroni, cheese, 160.
 Buttered cracker crumbs, 200.
 Whole wheat, bran or gluten bread.
 Arrowroot gruel, 205.
 Oatmeal gruel, 70.
 Barley gruel, 248.
 Barley gruel with broth, 308.
 Cracker gruel, 243.
 Flour gruel, 212.
 Farina gruel, 102.
 Indian meal gruel, 48.

 Fresh cream.
 Junket.
 Caudle, 404.
 Whey.
 Wine jelly, 165.
 Orange jelly, 150.
 Lemon jelly, 142.
 Grape jelly, 588.
 Peach jelly, 130.
 Cream jelly, 330.
 Spanish cream, 303.
 Bavarian cream, 205.
 Grape fluff, 150.
 Orange charlotte, 350.
 Strawberry moussé, 370.
 Jello Bavarian cream, 730.
 Soft custards, 192.
 Meringue, 110.
 Floating island, 506.
 Custard apple, 150.
 Custard orange, 150.
 Custard banana, 150.
 Custard peach, 150.
 Baked custard, 260.
 Omelet souffle, 230.
 Lemon souffle, 275.
 Custard souffle, 300.
 Peach meringue, 210.
 Junket, 300.
 Custard junket, 512.

 Corn starch pudding, 330.
 Pineapple cream, 340.
 Irish moss jelly, 677.
 Rice pudding, 746.
 Tapioca cream, 483.
 Apple tapioca, 110.
 Cracker pudding, 200.
 Any of the sherbets, 300.

 Fruit lemonade, 137.
 Bran lemonade, 20.
 Pineapple lemonade, 186.
 Irish moss lemonade, 25.
 Orangeade, 197.
 Fruitade, 45.
 Wine whey, 84.
 Malted milk and currant jelly, 85.
 Malted milk with wine, 88.
 Grape water, 135.
 Currant water, 15.
 Tamarind malted milk, 115.
 Egg broth, 319.
 Eggnog, 235.
 Egg and sherry, 388.
 Beef eggnog, 200.
 Egg and rum, 315.
 Egg and wine, 125.
 Egg lemonade, 192.
 Malted milk and egg, 120.
 Grape yolk, 150.
 Grape juice and egg, 270.
 Albuminized milk, 98.
 Albuminized grape juice, 40.
 Barley water, 180.
 Rice water, 160.
 Toast water, 350.
 Bread Panada, 162.
 Breakfast cocoa, 192.
 Malted milk cocoa, 83.
 Flaxseed tea.
 Butter, teaspoonful, 36.
 Butter and fermented milk.
 Malted milk.
 Rice milk, 458.
 Rum and milk, 186.
 Sherry or brandy and milk, 123.

Feedings should be at 8, 12, 4, and 8 o'clock, or three times a day with three supplemental feedings. The total values in the day should be (state the amount here) calories.

NOTE: These formulas may be found in the book entitled "Diet in Health and Disease," by Pattee. A. F. Pattee, Mt. Vernon, N. Y.

Most of the cases, however, are the ambulatory ones coming to the office. When the putrefaction is high they may be advised to use either one of the two following plans: In the first instance the diet is largely of fluid or semisolid type, foods are taken at 3 or 4 hour intervals during the day and evening and the regularity between them is preserved. The vegetables (none of the rough form), starches and cereals must be well boiled and mashed on the plate. The quantity taken at a time should be moderate and the foods eaten slowly. Meals should be begun at 8 A.M. and finished at 10 P.M. Most all foods are allowed.

Soups.

Split pea.	Vegetable soups.	Bisques.
Lentil soup.	Bouillon with egg.	Mock bisques.
Creamed soups.	Tomato bouillon with rice.	Victoria soup.

Eggs, Two a Day.

Soft boiled.	Poached on toast.	Omelette.
	Steamed or baked.	

Meat Substitutes.

Scrambled eggs, spinach, potatoes.	Nuts, macaroni, potatoes.	American or cream cheese.
Vegetable hash.	Oysters, any form.	Fried mushrooms on toast.

Combination of four vegetables

Chili con carne (Mexican style) with boiled rice.

Vegetables.

Boiled potatoes.	Beans and rice.	Buttered cabbage.
Riced potatoes.	Buttered beets.	Boiled carrots.
Mashed potatoes.	New England boiled dinner.	Spaghetti and tomatoes.
Creamed potatoes.	Stewed corn.	Mashed turnips.
Baked potatoes.	Spinach.	Lentils.
Spaghetti potatoes.	Stewed corn and rice.	Lima beans.
Boiled onions.	Rice and grated carrots.	Green peas.
Creamed onions.		Baked beans.

Cereals.

Shredded wheat.	Corn meal.	Hominy.
Rice and raisins.	Natural rice.	Barley gruel.
Rolled oats.	Corn meal mush, honey.	Sago and oatmeal gruel.
Gluten breakfast food.	Fruit and cereal.	Rice.
Whole wheat.	Cracker gruel.	

Fruits.

Raisins.	Grapes.	Oranges.
Fresh pineapple.	Prunes.	Baked apple.
Baked banana.	Compot.	Rhubarb.

Apple and preserved peaches.

Salads.

Fruit.	Sliced onion.	Ripe olives.
Fruit and nut.	Potato.	Tomato.
Vegetable combination.	Lettuce.	Romaine.
Mock chicken.	Celery.	Watercress.

Desserts.

Rice pudding.	Chocolate cake.	Chocolate pudding.
Bread pudding.	Banana cream cake.	Custards.
Raisin pudding.	Fruit charlotte.	Meringue.
Fruit pudding.	Honey.	Fruit whips.
Cereal pudding.	Fruit short cake.	Souffles.
Pies, fruit, graham crust.	Corn starch.	Junkets.
Gluten pudding.	Tapioca cream.	Gelatine.
Jellies.	Grape punch.	Fruit cream.

Beverages.

Cocoa.	Malted milk.	Yerba mate.
Milk, cold and hot.	Coffee hag.	Plain cool water.
Buttermilk.	Orange juice.	Fruit juice and water.
Orangeade.	Lemonade.	

Another plan is to use a diet essentially of the standard sort in which a large number of animal protein foods have been taken out. Of such a plan the following serves a useful purpose:

Fresh fruits (but no berries) are allowed with abundant fresh cream when possible.

Raw oysters and clams (no additions).

Vegetable broths, farinaceous broths, consommés and purées.

Any of the well cooked cereals served with milk, sugar and fresh cream

Eggs, in any form, only not more than two in a day.

Breads, rolls, biscuits, crackers and zwieback.

All foods made of gelatine.

Take a half pint of fresh cream and as much unsalted butter and olive oil during the day as possible.

All of the vegetables except potatoes, asparagus and canned goods, may be taken but they must be cooked to softness in a double boiler. Peas, beans, lentils, chicory and spinach are the most wholesome. The green vegetables and salads are allowed.

Custards, egg and milk, peeled fruits, jellies, marmalades, apples and pears.

In most instances, however, more or less protein foods may be taken and since it has been shown that the human being requires more or less protein for the metabolism of the body, I am inclined to the use of small quantities of meat, fish and chicken generally with these patients. Employing the standard form above, such an arrangement could be expressed in the following:

Fresh fruits peeled (but no berries) are allowed twice a day, with an abundance of fresh cream when possible.

Raw oysters and clams.

Vegetable broths, creamed soups and purées.

Any of the well cooked cereals allowed with cream and milk sugar.

Eggs any form, only two a day.

Breads, rolls, biscuits, crackers, baker's cake (flours specified).

Any form of gelatine foods.

Not more than three ounces of meat, fish or poultry in a day.

All of the vegetables excepting asparagus, potatoes and canned goods, unless home-made. The best vegetables are peas, beans, lentils, chicory and spinach. All the green vegetables and salads are allowed.

Farinaceous preparations, macaroni and porridges.

One-half pint of fresh cream and as much unsalted butter and olive oil as possible each day.

Desserts: Custards, apples (baked), cereal puddings, prunes, souffles, simple cakes of whole or cracked cereals.

Jellies: Apple, calves' foot, grape, lemon and berry juice.

Drinks: Water, cocoa and chocolate, made with cream and water.

In the definitely saccharo-butyric types the diet is largely that of protein, this being arranged in a practical way. For

this purpose I employ a high protein and green diet, somewhat as below, this being used for a short period of time largely to stimulate the proteolytic bacteria which usually are more or less inimical to those of the distinctly fermenting and acid types. After a short period of time, perhaps thirty days, the individual on this diet is shifted to a normal diet in which the large quantity of protein native foods is no longer important. Such a high protein diet could be constructed from the following plan, on which the patient should be instructed:

The diet is a temporary one. Take mostly meats—all forms of beef with the exception of cuts from the shoulder, kidneys and liver. The same pertains to lamb.

These meats should be fresh and taken in a broiled or roasted state. Mutton is permissible but no pork or veal.

Any kind of fish cooked as above, and to which boiled may be added, with the exception of shad roe and shell fish.

You may eat eggs in any form.

Any form of cooked, fresh vegetables that are green, such as spinach, beet tops, celery, etc., and any of the salads with French dressing very low in vinegar, may be taken.

Butter and whole milk are allowed, together with any form of simple cheese of the cream variety, such as Philadelphia and Neufchatel.

Eat as much of gelatine foods as possible.

Oatmeal and rolled oats are allowed.

Have breads or crackers made of gluten or rye flour.

Lentils and dried peas are permissible.

There is no objection to an occasional orange, pineapple or strawberries.

The best drink would be chocolate or cocoa.

All kinds of fermented milks may be taken—such as kumyss, matzoon, etc.

Later on the following foods may be added to the above diet:

Lettuce.	Tomatoes.	Grapefruit.
Cucumbers.	Brussels sprouts.	Lemons.
Spinach.	Water cress.	Butter nuts.
Rhubarb.	Sea kale.	Pignolias.
Endive.	Okra.	Unsweetened and un-
Marrow.	Cauliflower.	spiced pickles.
Sorrel.	Egg plant.	Clams.
Sauerkraut.	Cabbage.	Oysters.
Beet greens.	Radishes.	Scallops.
Dandelion greens.	Leeks.	Liver.
Swiss chard.	String beans.	Fish roe.
Celery.	Olives.	

In the mixed forms of toxemia the dietetic plan is distinctly different from the above. The idea now is to give food of the highest caloric value and smallest in bulk, these being of the most digestible type so as to permit of as little residue in the colon as possible. In these instances the diet may be of any plan selected providing the foregoing rules are observed.

The author here can offer no fixed plan of diet because it usually is required to make each one up specially for the individual. The main control of dieting in these instances is to allow only the smallest amounts of food that permit no loss of weight. After a study of the individual, taking all matters of age, sex, habitus, work, etc., into consideration, a diet should be arranged and the scales used daily to keep track of the weight. Such diets can be made up of various items in the foregoing plan, the first type of diet being the most useful in having the largest number of items.

As was stated before, too much dependence should not be placed upon diet in the treatment of intestinal toxemia. Some benefit can be brought about, of course, but the cure of the condition on the basis of altering the bacteriology by diet is only temporary, largely idealistic, and certainly not practically useful or permanent. A degree of benefit here and there can be brought about, but it is only transitory, because oft-times when a definite plan is followed (such as a low or full protein diet), after a course of time it is noted that the bacteriology present in the individual has now become facultative in the opposite direction and changed the type of toxemia from one form to another. In this sense it is not uncommon to see dieting doing more harm than good.

I also oppose the use of dietetic measures continuously, because, after a proper biologic diagnosis is made and biologic treatments are engaged in for the purpose of controlling the infection that exists in the intestine, opposite types of diet commonly interfere with results that are accomplishable. In this sense diet often interjects a factor difficult to estimate in a condition, which, both diagnostically and therapeutically, is complex enough even in its simplest phases. These are cases of an infection in the gut contents and the mucous membrane

of the small and large intestine, and for that reason no dieting could be of curative value.

Again, when a definite pathology is present no diet will avail distinctly, this also being so when there are errors of the secretions and the individual is toxic more from the mucous membrane than from the contents of the gut, and lastly when invasion has taken place mostly from an infection in the mucous membrane. However, the plans as mentioned above—the one in which the protein is low, the one in which the protein is high, and those based upon the study of the case in the nature of the process in the general biochemical way—are the most advisable that I know of; none of these, however, do I recommend too strongly.

Since it is important in all cases under biologic types of treatment that the best degree of bowel function and bowel elimination continue during the treatment, various additions to diet for the purpose of overcoming constipation or diarrhea are of value. It is my opinion that to a slight degree results of permanent cure in a case of intestinal toxemia are to some extent conditioned upon proper bowel function and movements. The statistics show that where the bowels have been obstinately constipated, the final result in the way of permanent cure has been modified. This modification, however, is not more than a matter of perhaps 10 per cent. of all the cases, but that 10 per cent. is of importance and I, therefore, am partial to the use of anticonstipation types of diet or anticonstipation additions to forms of diet, and to this end the various types of anticonstipation foodstuffs are employed.

A recent fallacy and fad in medicine is to standardize a diet on the *pH* value of urine, in which when the *pH* is below normal the individual is considered of the acid type and a so-called basic alkaline plan of dieting is employed. This newest fad, largely popularized by Sansum, is another instance of poor type of biologic bowel work and fails generally throughout the cases. However, a basic alkaline type of diet may at times serve to useful purpose in accomplishing good bowel elimination, and the type of diet which Sansum offers is represented in the following, the first being the so-called

basic alkaline plan, and the second being the normal plan. A better anticonstipation plan is on a subsequent page.

BASIC ALKALINE TYPE.

If the day is started with No. 1, finish the day out with No. 1. If with any other number, finish out with that number.

No. 1. **Breakfast:** Apple sauce, bacon, lima bean bread toast, butter, orange marmalade, glass of orange juice, glass of milk or cocoa.

Dinner: Browned potatoes, buttered carrots, Swiss chard, pineapple and cottage cheese salad with French dressing, lima bean bread, butter, fresh blackberries with cream, glass of orange juice, glass of milk.

Supper: Cream of spinach soup, baked potatoes, fresh asparagus, lima bean bread, butter, fresh apricots, glass of orange juice, glass of milk.

No. 2. **Breakfast:** Grapefruit, bacon, lima bean bread toast, butter, jelly, glass of orange juice, glass of milk or cocoa.

Dinner: Potatoes diced in cream, mashed turnips, buttered peas, head lettuce with French dressing, lima bean bread, butter, fresh raspberries with cream, glass of orange juice, glass of milk.

Supper: Cream of tomato soup, baked sweet potato, fresh spinach, lima bean muffins, butter, apple and cheese, glass of sweet orange juice, glass of milk.

No. 3. **Breakfast:** Baked apple, bacon, lima bean muffins, butter, cantaloupe, glass of orange juice, glass of milk or cocoa.

Dinner: Parsley potato, buttered string beans, Italian squash, lima bean bread, combination vegetable salad, butter, sliced peaches with cream, glass of orange juice, glass of milk.

Supper: Cream of celery soup, scalloped potatoes, diced beets, lima bean bread, butter, fresh cherries, glass of orange juice, glass of milk.

No. 4. **Breakfast:** Banana with cream, bacon, lima bean bread toast, butter, apple sauce, glass of orange juice, glass of milk or cocoa.

Dinner: Cream of pea soup, whipped potatoes, baked squash, buttered celery, peach basket salad, lima bean bread, butter, maple nut sundæ, glass of orange juice, glass of milk.

Supper: Cream of asparagus soup, buttered carrots and peas, creamed turnips, sliced tomatoes, lima bean bread, butter, ripe pear, glass of orange juice, glass of milk.

No. 5. **Breakfast:** Cantaloupe, bacon, lima bean muffins, butter, stewed raisins, glass of orange juice, glass of milk or cocoa.

Dinner: Split pea soup, candied sweet potatoes, purée beets, fresh spinach, lima bean bread, butter, frozen apricots, glass of orange juice, glass of milk.

Supper: Creamed potatoes, buttered string beans, banana and orange salad, lima bean bread, butter, grapes, glass of orange juice, glass of milk.

No. 6. **Breakfast:** Fresh figs, lima bean bread toast, butter, strawberry jam, glass of orange juice, glass of milk or cocoa.

Dinner: Cream of celery soup, scalloped potatoes, baby lima beans, Swiss chard, pickled beet salad, lima bean bread, butter, sliced peaches with cream, glass of orange juice, glass of milk.

Supper: Cream of carrot soup, buttered peas, banana squash, pineapple salad, lima bean bread, butter, junket, nuts, glass of orange juice and milk.

No. 7. **Breakfast:** Baked apple, bacon, lima bean bread toasted, butter, grapes, glass of orange juice, glass of milk or cocoa.

Dinner: Cream of string bean soup, baked potato, creamed carrots, buttered fresh asparagus, tomato salad with French dressing, lima bean bread, butter, honey dew melon, cluster raisins, glass of orange juice, glass of milk.

Supper: Potato balls with parsley sauce, creamed salsify, spinach, lima bean muffins, butter, apricot charlotte, glass of orange juice, glass of milk.

No. 8. **Breakfast:** Fresh raspberries with cream, bacon, currant jelly, lima bean bread toast, butter, glass of orange juice, glass of milk or cocoa.

Dinner: Cream of cauliflower soup, mashed potatoes, artichoke, beet greens, combination fruit salad, lima bean bread, butter, apple tapioca, glass of orange juice, glass of milk.

Supper: Stuffed potatoes, baked tomatoes, buttered string beans, lima bean bread, butter, blackberries, glass of orange juice, glass of milk.

No. 9. **Breakfast:** Sliced peaches, bacon, lima bean muffins, butter, raspberry jam, glass of orange juice, glass of milk or cocoa.

Dinner: Cream of onion soup, browned potatoes, creamed cauliflower, parsnips, Waldorf salad, lima bean bread, butter, pineapple ice, glass of orange juice, glass of milk.

Supper: Potatoes au gratin, buttered string beans, tomato and cottage cheese salad, lima bean bread, butter, canned figs, glass of orange juice, glass of milk.

No. 10. **Breakfast:** Fresh strawberries, bacon, lima bean bread, butter, cherry preserves, glass of orange juice, glass of milk or cocoa.

Dinner: Mashed potatoes, Brussels sprouts, mushrooms, chicory salad, lima bean bread, butter, celery, glass of orange juice, glass of milk, fresh apple with cheese.

Supper: Cream of celery soup, French fried potatoes, broiled onions, lima bean muffins, butter, iced peaches, glass of orange juice, glass of milk.

Lima bean flour may be secured from the California Lima Bean Growers Association of Oxnard, California, and they will furnish recipes for cooking. Lima bean flour may safely be mixed with ordinary flour, but the latter should not be eaten alone, it being too acid. In correcting your trouble by means of diet, specimens of urine require to be examined from time to time. These must be voided just before coming to the office and brought in with you, or best voided here.

BASIC NORMAL TYPE.

This diet is balanced throughout. If you start the day with No. 1 follow No. 1 through that day, and if any other number follow that through the day. If any special food is suggested which disagrees with you omit it but do not substitute another in its place (except in the case of fruits or vegetables).

No. 1. **Breakfast:** Melon, shredded wheat with milk and sugar, scrambled eggs, toast and butter, coffee with cream and sugar, orange juice.

Dinner: Tomato bisque, roast chicken with dressing, baked squash, mashed potato and gravy, buttered celery, pineapple and marshmallow salad with whipped cream dressing, bread and butter, chocolate sundæ, cake, tea with cream and sugar.

Supper: Creamed macaroni, spinach on toast with poached egg, baked tomato, bread and butter, assorted fresh fruits, milk.

No. 2. **Breakfast:** Orange sections, oatmeal with milk and sugar, Melba toast, butter, bacon, coffee with cream and sugar, grapefruit juice with lemon.

Dinner: Vegetable soup, breaded veal chop, creamed cauliflower, scalloped potatoes, small peas, tomato salad with French dressing, bread and butter, apple Betty with hard sauce, tea with cream and sugar.

Supper: Liver and bacon, baked potato, buttered asparagus, bread and butter, baked apple, milk.

No. 3. **Breakfast:** Banana, corn flakes with milk and sugar, coddled egg, toast and butter, coffee with cream and sugar, orange juice.

Dinner: Cream of celery soup, roast beef, buttered carrots, browned potatoes, stewed tomatoes, Waldorf salad, bread and butter, cottage pudding with strawberry sauce, tea with sugar.

Supper: Creamed chicken on toast points, baked sweet potato, baby lima beans, bread and butter, milk.

No. 4. **Breakfast:** Apple sauce, cream of wheat with milk and sugar, bacon, toast and butter, coffee with cream and sugar, orange juice.

Dinner: Melon, sirloin steak, string beans, mashed brown potatoes, eggplant, pear and cottage cheese salad with French dressing, bread and butter, fruit jello, tea with cream and sugar.

Supper: Stuffed potato, scalloped cabbage with cheese, egg ring salad with boiled dressing, bread and butter, hermits, fresh sliced peaches. Milk.

No. 5. **Breakfast:** Sliced peaches, wheatena with milk and sugar, omelet with currant jelly, wheat muffins and butter, coffee with cream and sugar, orange juice.

Dinner: Vegetable soup, roast lamb with mint sauce, buttered onions, mashed potatoes, small peas, orange and grape salad with French dressing, bread and butter, ginger bread with whipped cream, tea with sugar.

Supper: Braised sweetbreads with mushroom sauce, cream potato, buttered Italian squash, bread and butter, red raspberries, sugar cookies, milk.

No. 6. **Breakfast:** Stewed prunes, puffed rice with milk and sugar, poached egg on toast, Melba toast, butter, coffee with cream and sugar, orange juice.

Dinner: Cream of corn soup, baked halibut, parsley potato, sliced tomato, lettuce salad with French dressing, fruit sherbet, wafers, tea with cream and sugar.

Sugar: Cheese souffle, baked potato, fresh spinach, combination vegetable salad with boiled dressing, bread and butter, fresh strawberries, milk.

No. 7. **Breakfast:** Melon, pettjohn with milk and sugar, bacon, toast and butter, orange juice.

Dinner: Split pea soup, lamb stew with vegetables, boiled potato, stuffed prune salad with cottage cheese and whipped cream, bread pudding with fruit sauce, tea and sugar.

Supper: Baked beans, Spanish pickles, fresh spinach, orange and grape salad with French dressing, corn bread, butter, baked apple, milk.

No. 8. **Breakfast:** Grapefruit, shredded wheat with milk and sugar, scrambled egg, toast and butter, coffee with cream and sugar, orange juice.

Dinner: Fresh fruit cocktail, chicken broth with rice, broiled chicken, mashed potatoes and gravy, buttered asparagus, celery hearts, small peas, olives, pineapple sundæ and cake, coffee and sugar.

Supper: Boiled rice, string beans, baked tomato, tuna salad, hot biscuits and butter, apricot whip, milk.

No. 9. **Breakfast:** Rhubarb, cream of wheat with milk and sugar, liver and bacon, toast, butter, coffee with cream and sugar. Orange juice.

Dinner: Vegetable soup, roast beef, potato cake, buttered celery, cold slaw, creamed carrots, bread and butter, stuffed baked apple, tea with cream and sugar.

Supper: Jelly omelet, buttered peas, braised lettuce, pineapple and cottage cheese salad with French dressing, bread and butter, peach compote, milk.

No. 10. **Breakfast:** Orange, shredded wheat with milk and sugar, broiled ham, toast with butter and jelly, grapefruit with lemon, coffee with cream and sugar.

Dinner: Cream of celery soup, Swiss steak, baked sweet potato, sliced tomatoes, pear and American cheese salad with French dressing, bread and butter, tapioca cream, tea with sugar.

Supper: Chicken soufflé with mushroom sauce, baked potato, egg plant, asparagus salad with French dressing, bread and butter, sponge cake, milk.

CONSTIPATION DIETS.

If constipation exists or ensues, in place of or in addition to the fruit juices listed, another fruit or vegetable must be added to each meal. In my experience and observation, it serves to better purpose to use one of the other plans of diet recommended and to add to whatever plan is used one or more of constipation items. Briefly expressed, such a plan could be as follows:

Take sufficient out-door exercise each day. Rest for fifteen minutes before meals and a whole hour prone after them.

Eat at regular hours in a quiet place. The atmosphere at the table should be congenial.

Drink only between the main meals or at the supplemental ones.

Fireless cook or steam vegetables, cereals and grains.

Use cracked corn and whole wheat and not plain corn meal nor plain white flour. Use all the grains in their natural state, cracked or ground, as far as possible. Use the natural form of rice not the usual polished kind.

Instead of cane or table sugar (which is constipating) use as sweetening, lactose (milk sugar), honey, molasses or maltose granulated or syrup.

Partake abundantly of ripe fresh fruits, vegetables, greens, spinach, chicory, raisins, grapes, tomatoes (raw), carrots, orange juice and egg yolk. Eat cabbage if tolerated, sprouts, cauliflower, oatmeal; but nothing containing pits or seeds.

Do not drink milk, tea or coffee. In their place drink fresh cream and water, Paraguay tea or plain water.

Eat plentifully of bran foods, formulas of them enclosed in package.

Drink five glasses of water each day, always when the stomach is empty.

Measures to encourage the bowels to move in a normal way are the following:

Begin by taking three of the bran gems during the course of the day.

If you cannot get them, you may take in place of them a good-sized dish of pettijohn bran, or from 1 to 2 tablespoonfuls of wheat bran mixed with oatmeal or shredded wheat biscuit for breakfast, or one of the other bran combinations.

If the above does not move the bowels, retain the bran method which you are employing and as an additional method take a dish of stewed prunes cooked with milk sugar, honey or molasses, at the evening meal or before retiring.

If the foregoing two methods are not efficient, take the prunes at the evening meal and a tablespoonful of petroleum jelly or vaseline before retiring. With the institution of this, you are to take from three to six glasses of water during the day, in the intervals between meals, on arising and before retiring.

If then the bowels do not move, add a few senna leaves to the prunes which are taken, these being put in at the time of stewing.

If still the bowels do not move, drop the use of the bran both in the biscuits and at breakfast and substitute a handful or one-half ounce of agar-agar (finely cut) eaten with cream and milk sugar for breakfast. This may be mixed with the cereal but not cooked with it.

The foregoing methods should be carried out by beginning with the first and adding one thing after the other until the desired result is obtained.

Do not become alarmed if the bowels do not move in the beginning, or if a day goes by without a movement during the time you are on the above additions.

At certain times each morning and evening, an effort should be made to have the bowels move. Persist in sitting ten minutes even if not successful. During this time the feet should be raised from the floor by a foot stool, box or otherwise, so that the knees are raised above the hip level.

CONSTRUCTIVE TYPE OF DIET.

Occasionally it serves to purpose, in individuals who are distinctly below par physically and vitally, to add to the diet certain measures of constructive type. To any plan of diet

the following may be added, since it occasionally serves to worth-while purpose:

Take out-door exercise before meals, and a rest of fifteen minutes before them and a whole hour prone after them.

Eat slowly, cut the foods fine and masticate well.

When possible start the meal with a slice or two of dry toast, or some other form of dry food.

Use grains and cereals in their natural state, ground or cracked, instead of that found in many forms of biscuits, refined cereals (ready forms of breakfast foods), white bread and the usual baker's cake, rolls, etc. Do not eat pearled barley, corn starch, egg white and cane sugar.

When allowed to be taken, the meat, fish and fowl should always be roasted, broiled or baked.

Take all the gelatine foods desired—these may be flavored with mint, orange, lemon, pineapple, cherry, loganberry and strawberry.

All salads with abundance of Italian olive oil may be taken.

Drink only cream and water (never milk), malt and cream, cocoa and chocolate made of cream and water instead of milk.

Drink only between the main meals or at the supplemental meals.

Eat nuts if desired and use bran as much as possible.

Change the taste of the same foods by different cookery as much as possible.

Do not eat hashed, made-up dishes, foods that are kept over from day to day. Whenever possible get the foods direct from producers instead of from the local markets.

It must now be apparent to the reader that the plans of diet followed are simply general plans which are standardized on the gross classification of toxemic types and that these should not be depended upon to control the bacterial side of the case, or if so for not more than a short time. Fussing around with diets, changing them here and there in various food selections, never has cured a case of well-established chronic intestinal toxemia of the primary type, because the important part of this work is an accurate biological diagnosis and therapy entirely dependent on the biologic handling of the condition.

CHAPTER XIII.

ADDITIONAL MEASURES OF TREATMENT.

While the handling of intestinal toxemia is practically altogether a biologic affair, and to a very limited extent one of dietetics, additional measures of treatment have been found useful in my work. These recorded briefly are as follows:

Fresh Air and Exercise.—Fresh air and exercise are beneficial in the ways that have been stated in the chapter on environments. Even though individuals after the fortieth year of age do not require as much exercise to keep fit as those who are younger, nevertheless a certain amount of exercise is necessary to maintain proper metabolism, especially in the large part of the human body that is made up of the voluntary muscles. Sometimes this can be accomplished by the use of any of the special apparatuses for the purpose, the most commonly used being some form of weight-pulling or elastic-stretching apparatus, usually attached to a wall. The limitation of these is that whereas the upper part of the body is quite well exercised, that of the abdominal muscles and the lower extremities is not. For this reason the setting-up exercises which are used as a part of the military drill of any of the governments serve to good purpose, although all are deficient in that they do not require the individual to exercise the abdominal muscles properly.

Whatever the form of apparatus used may be, it is required for these people to do considerable work on their backs on the floor in the form of any of the exercises which bring the abdominal muscles strongly into play, because in the adult human being, the most important part of the body to exercise is the abdominal muscles, the ones more often neglected than any other. The raising of the lower extremities from the floor or the raising of weights placed upon the abdomen serves to good purpose. While the average woman in the home thinks she gets enough exercise in her domestic work, this is rarely

the case, the same being true with the business man who rushes to his office and back again after his hours of work have ceased. The equivalent of several miles of walking a day, especially if it is accompanied with deep breathing, is quite excellent.

Any form of exercise which is done in the fresh air not only burns up the deficiently oxidized substances in the body, but so increases the oxygen content of the tissues, and helps to burn and bind up the toxic material in the blood. It is this factor of fresh air, in addition to sunshine and some exercise, that very largely results in benefits in these people when they sojourn in the country or repair for a few weeks to some sanitarium. By exercise, sunshine and fresh air, one gets, so to speak, on top of the toxic materials in the blood and in that way the tissues are saved for the time being.

Sunshine.—Sunshine, especially when the entire surface of the body can be exposed, causes opsonin development (which opsonins have an immunizing effect) and the stimulation of the leukocytes, especially those of the phagocytic type, this acting directly in an antitoxic way. With those who cannot for business and other reasons leave the city at various times during the year, who are closed up in their homes, or in their offices, a short sojourn in the country at week-ends, the playing of golf, and the use of the quartz lamp, perhaps, during the winter months—all serve to good purpose.

Rest and Food Cures.—Not a few of these people are definitely debilitated, others have irritative states of nervous system, and others again have acquired such bad routines of life that a rest and food cure for a time would be helpful. This is conducted in the usual ways of such procedures and in my experience serves well for the acquiring of warm specimens of stool for bacteriologic investigation. Where a neurologic element is a factor, such as in spastic states of the gastroenteric canal, states of hypersensibility, emotional amenities, and where routine cannot be followed, it is wise that such rest and food cures be conducted in a sanitarium in which the environment and atmosphere are conducive to the best results. After the patient is permitted to return home it sometimes is

wise to prescribe a daily routine in which the features of rest are incorporated, such as remaining in bed until the noon meal, or remaining on the back in a prone position for one hour after each meal, and the cutting out of entertainment and excitement which deprive them of sufficient hours of sleep. In this connection, even in the marked insomnia cases, regular hours for sleep should be insisted upon and the patients told that even if they do not sleep they are resting and being benefited just the same.

Baths.—Since the elimination of some of the toxic substances from the intestines is by way of the skin, several hot baths a week serve to keep the skin active and enhance perspiration. In a number of these people the skin is dry, perspiration is difficult, and only by prolonged immersion followed by the drying of the body in hot blankets, or the use of hot packs, can more or less perspiration be excited. I am not partial to the use of cold baths for these people. They accomplish nothing in increasing vigor, the majority of these patients do not react favorably, and very often cold baths are the cause of bringing on various neuritic types of pain. Instead of them, I am partial to the use of warm baths, preferably at night before retiring, and where there exists more or less nervousness, a pine-needle extract preparation being used in the water, the individual remaining in the bath long enough to accomplish relaxation and subsequent sleep. During the hot bath, the imbibing of draughts of cool water is sometimes of benefit.

In addition to the above it sometimes serves to good purpose to wrap these people up in an electric blanket two or three times a week so as to cause an active perspiration. Such sweatings are especially useful in cases of kidney irritation and involvement, and in the menopausal woman.

Endocrine Therapy.—Since many of these individuals are vagotonic, endocrine therapy is sometimes of value in a symptomatic way. The most important is the use of small doses of thyroid substance to which the other endocrine substances can be added, and the use of lecithin and various high vitamin-bearing substances. It is not infrequent that small doses of luminal are beneficial in capsules containing the

various glandular preparations. As has been stated in a former chapter, more important than the use of the endocrine substances is to remove the fundamental intestinal toxemia and allow the glands to resume their normal function.

Tonics.—Of course in the various anemias more or less tonic measures are required. Of these I am more partial to the use of the glycerophosphate preparations containing a small amount of strychnine and given hypodermically than any other. However, there are a number of substances, especially those of organic iron preparation, that are most helpful. In this connection it may be mentioned that other types of intravenous and subcutaneous medications are required in cases here and there, all of which can be carried out while the biologic treatments are being employed. Occasionally any of the tonics, especially some of the proprietary types that are more easily assimilated and cause less harmful effects upon gastric digestion as well as constipation than the old mixtures and remedies, serve to good purpose. It will be noted that I am not recommending the use of any medicinal substance to benefit an intestinal toxemia. My reason is that, after an expensive trial of them, they all have been found to be practically one hundred per cent. useless.

Other Constructive Measures.—Of much value, however, here and there, is the use of various constructive measures. In the saccharo-butyric types of intestinal toxemia where obesity exists as one of its symptoms, naturally constructive measures are not required. But in many toxic persons the general running vitality is at a lowered level and more or less constructive bucking-up is called for. In this connection considerable can be accomplished by the use of regulating diets on a high vitamin basis, and the general use of tomato or orange juice taken raw or cooked and small doses of cod-liver oil after meals. It should be mentioned here in connection with cod-liver oil that one receives all the vitamin A and D necessary in doses as small as 4 c.c. (1 teaspoonful) three times a day.

Abundant feeding even in the face of distressing gastric symptoms must often be insisted upon, and smaller quantities

of food taken at a meal, meals taken five or six times a day often being most valuable. In this same connection hydrocarbons should be forced, especially fresh cream, butter and olive oil. It is best here to state definitely to the patient how much of these substances must be taken in the course of the day rather than to leave it to his selection. The iron-bearing vegetables are also of use as is the employment of liver and bone marrow. While the author has employed many ways of using liver and the different extracts on the market, his practice today is to specify the amount of raw liver required to be taken in the course of the day, to have this ground up and cooked in soup, perhaps with vegetables or some cereal. This is more pleasant to take than any form of liver in its raw state, and the cooking of liver does not destroy much of its hematopoietic properties. Of course raw or nearly raw liver would be best.

Marrow bones cracked and baked in the oven, the marrow scraped out and put on moist toast and baked again is an excellent constructive food measure. Not infrequently the use of malt preparations, either in the form of extracts of malt or the abundant use of maltose sugar is to be desired. There is no better therapeutic remedy for the neurotic or nervous individual than the long-continued use of some form of malt preparation. Yeast in various forms has been employed, particularly in connection with staphylococcic toxemias, rather for their vitamin content and never for any benefit on an intestinal toxemia. I have followed several hundred cases of primary intestinal toxemia where yeast was being taken and I have yet to see the first biologic benefit from its use.

Elimination.—Lastly it should be mentioned that the more active the elimination (other than by way of purgation) the less the status of toxemia in the body outside of the intestinal canal. To this end fresh air, increasing the body oxydase, and inducing better elimination in the expired air, attention to the skin which has been mentioned, and the drinking of sufficient fluid to cause increased elimination by the kidneys are all worth while. It is best that these fluids be in the form of water taken before meals, and there should

be at least five glasses of water taken in the course of twenty-four hours. As to elimination by purgation, the rule is that the less the use of purgation substances the less the degree of toxemia, and the more the use of purgations the greater the toxemia and the difficulty of its control. To this end, however, it occasionally is necessary to use some very small doses of cascara by mouth, or better yet, glycerine suppositories or small tap water enemas with table salt in the evening before retiring if the bowels have not moved during the course of the day. Especially is this useful when anaphylactic phenomena are imminent or present from biologic treatments.

It may be emphasized here that there is no more definite way to defeat any method of biologic treatment in the intestinal canal than to allow the individual to take purgative remedies by mouth in any steady way. I have almost gotten to feel that if the bowels cannot be regulated by diet or most simple means, one's chances of accomplishing a sustained beneficial effect by biologic treatment begin to be modified, and here and there (10 per cent.) are certainly defeated if constant purgation is engaged in. On the other hand, not uncommonly in the beginning of biologic treatment some degree of assistance is required, and when the gut reestablishes itself on a more normal biologic and chemical basis, proper bowel function without any assistance commonly is accomplished. It is my opinion that the commonest cause of chronic constipation in the human being is found in the states of intestinal toxemia which have caused pathologic changes in the colon and various degrees of a dry colitis. And in this I am satisfied that when the diagnosis is accurate, especially the biologic one, and when the biologic methods selected are accurate in causing the desired changes to be brought about, that the colon reestablishes itself approximately to normal, and when it does, normal bowel function is the rule in as high as 95 per cent. of instances.

CHAPTER XIV.

QUESTIONNAIRES TO THE PROFESSION.

The author's experience in results from the treatment of chronic intestinal toxic states covers a wide range of medical subjects. Many of the patients had been referred for the diagnosis and treatment of some disorder in the digestive tract, the majority intestinal in location, by specialists belonging to one of the major or minor departments of medicine. Generally these individuals at the time were under treatment by these different specialists for some condition encompassed in their field of work. Ofttimes the benefits accrued from the treatment of some toxic intestinal condition caused a remarkable result in the condition handled by the specialist. Not infrequently much comment and interest was brought about by these results, so much so that many claimed that the intestinal state was directly responsible or contributory in the production of the disorders treated by them. Since the cases were referred by specialists in all of the branches of medicine, opportunity was given to observe results in a wide variety of diseases and disorders. Therefore, as will be presented, efforts are made to study larger groups of these handled by many of the specialists in the different fields.

To acquire information and the viewpoints of the different specialists on intestinal factors considered important in diseases and disorders in their fields of work, questionnaires were distributed among the most prominent specialists in the United States, the British Empire and the European countries. The vast majority of these were sent to those throughout the United States. Largely they were sent to the more prominent and well-known members of the different national societies devoted to study of subjects in the different specialties. Also included were those who occupied teaching chairs in the specialties in the various medical schools, the men who had charge of these departments in most of the well-known clinics, to-

gether with those known by reputation to be most capable in their work. The replies to these questionnaires were compiled and will be presented in the entity terms as sent in. Before offering this, however, some interesting side-lights on these are worthy of note.

Specialists in the same field commonly use different terms to designate the same condition. Ofttimes these terms are so confusing as to tend dangerously to lead the reader of literature in which they occur to unnecessary doubts as to what they represent. Especially is this true in neuropsychiatry and neurology, ophthalmology, otorhinolaryngology, and orthopedics, although no department of medicine seems free from them and to have a standard nomenclature of entity terms. One would be inclined to make the suggestion that committees on nomenclature should be established by the national organizations to help bring this desirable result about, and that efforts should be encouraged to wipe out the old and synonymous types of terms and to discourage new ones springing up here and there. As many of these now in usage are unnecessarily confusing, except perhaps to the most erudite specialist, if the matter were settled in agreeing on one or perhaps two terms it would be less burdensome to the younger men developing in these specialties and the average medical reader.

As was expected, the most diverse attitudes were presented from equally prominent men in the same specialty as to the importance of chronic intestinal toxemias in connection with conditions in their field. A few such personal opinions are presented in connection with the lists. They ranged all the way from "None at all," "Maybe," "Have had no experience with a connection," "The above are definitely due to intestinal conditions, others not so," to "The intestinal canal is the one big factor in the production of the diseases in our work." Others in the same field tried to make distinctions in groups of diseases such as "The acute are not, the chronic perhaps," "The acute sometimes, and the chronic practically all of them," "They are found as causes in the acute as well as the chronic." With some of the men the idea of such a connection was a "new one," and others an "old one" which had received their

attention for years and because of these attentions they felt the benefits accomplished in their work were "slightly," "distinctly," "generally," or "markedly" enhanced.

Test cases of one entity here and there in different departments that had received the largest number of positive answers in an etiological sense were made and definite questions were asked on it of thirty prominent men in the one specialty, and there were as high as twenty-nine different opinions in instances which ranged all the way from pro to con, and this diversity of opinions was general in all the specialties.

Quite generally it was proven that none of the men or in any of the medical institutions or clinics of this country was worthwhile work being done on the intestinal biologic disorders (and of course on their treatments). Mostly no effort of any kind was made, even though numerous men in these institutions believed in a primary or contributing connection. The questionnaires to heads of laboratories brought an occasional interest in a Gram-differential stained count of a cold specimen of stool (of course blood, excess fats, etc., were of interest), but no deductions, interest or work beyond this, in either stools, urines or blood. In toto, the questionnaires proved that the most common disorder of man and the most potent producer of disease, neither in diagnoses nor therapeutics, was being paid any attention to, and if so, it was largely on an assumption type of diagnosis on the part of the clinicians and with treatments quite as deficient.

The results of the questionnaires are herewith presented, the duplicating terms being omitted as far as possible. Those terms occurring at the beginnings of the lists were the most often mentioned. The more conspicuous of the general statements made are subnoted to each list. Those more or less similar to the ones presented were omitted for brevity's sake or allocated to one of those mentioned.

INTERNAL MEDICINE.

Anemia, primary and secondary.	Pyelitis.
Hepatitis, acute and chronic.	Cystitis.
General debility (vital and physical).	Urticaria.
Arteriosclerosis.	Vasomotor irritability.

INTERNAL MEDICINE (*Continued*).

Chronic arthritis.	Appendicitis.
Psychasthenia.	Epilepsy.
Hypertension.	Neuritis.
Neurasthenia.	Gastric disturbance.
Acute nephritis.	Pellagra.
Chronic nephritis.	Sprue.
Chronic constipation.	Obesity.
Migraine.	Cardiovascular-renal disease.
Hyperchlorhydria.	Neurosis.
Gall-bladder disease.	Psychosis.
Intestinal tuberculosis.	Chronic colitis.
Hirschsprung's disease.	Indigestion.
Celiac disease.	Hyperthyroidism.
General toxemia (cecal stasis).	Angina pectoris.
Indicanuria.	Endocarditis (acute and chronic).
Periodic headache (due to cecal stasis).	Thyroiditis (acute and chronic).
Toxic headaches.	Pneumonitis (acute and chronic).
Cardiac irregularities.	Myocarditis (acute and chronic).
Cholecystitis.	Mental states (acute and chronic).
	Blood dyscrasias (chronic).

"Directly or indirectly the whole field of medicine."

"How can one tell?"

GASTRO-ENTEROLOGY.

Gall-bladder infections.	Redundant colon.
Arthritis (some forms, not stated).	Atonic colon.
Appendicitis.	Spastic colon.
Pancreatitis.	Hyperthyroid states.
Intestinal stasis.	Hypothyroid states.
Hepatitis (and other disorders of the liver).	Sphincter spasm.
Achylia gastrica.	Sphincter incompetence.
Psychasthenia.	Spastic recto-sigmoid apparatus.
Dermatological conditions.	Headaches.
Neuroses, sensory.	Eye diseases.
Neurasthenia.	Throat conditions.
Functional cardiac disturbances (palpitation pains).	Kidney disorders.
Migraine.	Pernicious anemia.
Pyelitis.	Liver and biliary tract disease.
Ulcer (gastric and duodenal).	Catarrhal jaundice.
Erosions of stomach.	Diabetes mellitus.
Gastro-intestinal atrophy.	Arthritis.
Gastro-enteroptosis.	Gastric disturbance.
Colitis, acute and chronic.	Focal infections.
Diverticulosis and diverticulitis.	Anemia, secondary.
	Renal disease.
	Vertigo.

GASTRO-ENTEROLOGY (*Continued*).

Pyloric obstruction.	Upper respiratory tract disease
Gastritis, chronic.	(chronic).
Ileocecal incompetency.	Circulatory derangement.
Some diseases of the rectum.	Myalgia.
Angioneurotic edema.	Neuralgia.
Ulcerative colitis.	Sprue.
Catarrhal colitis.	Pellagra.

"Practically important in all diseases."

"Only interest the toxemia of acute or chronic intestinal obstructions and those of potential obstruction of ileus."

"Doubt that such exists."

CARDIOLOGY.

Premature beats (contractions, auricular, nodal, ventricular).
 Paroxysmal tachycardia.
 Angina pectoris (in relation to gastro-intestinal gas).
 Extrasystoles (due to gall-bladder and appendix disease).
 Hypertensive heart disease.
 Chronic mitral disease (with cardiac liver and fermentation).
 Achlorhydria.
 Mucous colitis.
 Coronary sclerosis.
 Tachycardia (simple acceleration).
 Hypertension.

"None in relation to intestinal toxemias."

"With our lack of knowledge unfortunate that connection is considered."

"The 90 per cent. factor of the whole field of heart disease."

NEUROPSYCHIATRY AND NEUROLOGY.

Epilepsy.	Depression, cyclothymic.
Neurasthenia.	Menopausal neuroses.
Anxiety states.	Gastric tetany.
Migraine.	Irritable colon.
Psychoneurosis.	Funicular myelitis (from pernicious anemia).
Combined cord degeneration.	Cord changes from intestinal parasites.
Neuroses (intestinal types).	Cord changes in pellagra.
Neuroses (all of them).	Peripheral cord changes.
Colitis.	Post encephalitis.
Manic depressive psychosis.	Depression psychosis.
Psychoses (all the functional ones).	
Gastro-intestinal-rectal crises.	
Depression, simple reactional.	

"No knowledge of any connection between intestinal toxemia and neuropsychiatric diseases."

"Many of nervous disorders are primary or secondary from intestinal disorders."

"The most important of all factors."

ORTHOPEDICS.

Arthritis (all types, including atrophic and hypertrophic types).

Arthritis (the osteo-arthritic type).

Spinal arthritis.

Infective arthritis (rheumatoid).

Neuritis.

Myositis.

Postural defects (adults).

Constipation in children posture problems.

Constipation in muscle injuries.

Myo-fascitis	{	sacro-iliac strain or relaxation.
		sciatica.
		pes planus.
		acute or chronic lumbago.
		sciatic scoliosis.
		tennis elbow.

Various joint conditions (notably hip and knee).

Sciatic scoliosis.

Vague joint disturbances.

General debilities.

Low back strain (sacro-iliac strain).

Mistaken diagnoses (sciatica, neuritis, lumbago).

"Very important."

"The better the physician, the better his orthopedic handling."

"I'd like to know."

OTORHINOLARYNGOLOGY.

Sinus disease (acute or chronic suppurative).

Tonsillitis (acute and chronic).

Rhinitis hyperaesthetica.

Rhinitis hyperplastic.

Tonsillitis, purulent.

Chronic laryngitis.

Chronic tracheitis.

Coryza (common colds).

Progressive deafness.

Chronic nasal sinusitis.

Rhinorrhea (edema of nasal mucosa).

Tonsillitis (chronic).

Rhinitis, chronic irritative.

Tonsillitis, follicular.

Vertigo.

Perennial hay-fever.

OTORHINOLARYNGOLOGY (*Continued*).

- Many types of chronic deafness.
- Certain types of deafness.
- Sinus disease (significant symptoms).
- Lymphatic engorgement of nasopharynx (due to lack of fat vitamins).
- Labyrinthine disturbance (due to intestinal stasis).
- Asthma.
- Polyp formations (nasal and sinus).
- Nerve tympanic deafness.
- Middle ear disease (stubborn cases).
- Eustachitis (stubborn cases).
- Infants { middle ear.
- mastoid.
- sinus infections.
- Vasomotor rhinitis.
- Chronic rhinitis.
- Nasopharyngeal obstructions.
- Otitis media (chronic suppurative).
- Chronic pharyngitis.
- Headaches.
- Non-suppurative tubotympanic "catarrh" without fixation.
- Spasmodic croup in children.
- Bronchial asthma.
- Acute catarrhal otitis media (in diarrheic or epidemic forms).

"Intestinal canal most important source of diseases and conditions."

"Practically all that come into this field of medicine."

"The list of diseases and disorders too long to tabulate."

"In all chronic inflammatory conditions, intestinal toxemia is the rule rather than the exception."

"Practically the complete list of all nose and throat conditions."

"All stubborn or severe nose and throat conditions."

OPHTHALMOLOGY.

- | | |
|-----------------------------------|---|
| Chronic uveitis. | Vertigo. |
| Iritis. | Chronic blepharitis. |
| Choroiditis. | Asthenopia. |
| Conjunctivitis (chronic, dry). | Vitreous opacities. |
| Keratitis (superficial and deep). | Herpes corneæ. |
| Choroidoretinitis. | Scleritis. |
| Paracentral color scotoma. | Keratitis (marginal). |
| Scintillating scotoma. | Neuroretinitis. |
| Shifting toxic scotoma. | Glaucoma (perhaps). |
| Cataract. | Fungacious (gouty) episcleral, conjunctival infections. |
| Retinal disease. | Simple asthenopia (with headaches after use of eyes). |
| Presbyopia. | |
| Relapsing iritis. | |

OPHTHALMOLOGY (*Continued*).

Endogenous intraocular infections (postoperative).	Optic neuritis (mild). Arteriosclerotic retinal changes.
Choroiditis (low grade, floating opacities of vitreous).	Retinitis (albuminuric and diabetic).

"Have observed no connection between intestinal toxemia and the eye."

"Not to much extent."

"In many conditions."

"Only a possible factor."

"The most important factor in functional and chronic eye diseases."

"Much stress should be laid on intestinal conditions causing eye diseases."

DERMATOLOGY.

Urticaria (recurrent).	Pityriasis rosea.
Eczema (acute and chronic).	Acne indurata.
Erythema multiforme.	Neurodermatitis.
Acne rosacea.	Pellagra.
Psoriasis.	Prurigo.
Acne vulgaris.	Diabetic dermatosis.
Pruritus.	Inflammatory dermatosis (in- directly).
Angioneurotic edema.	Erythema scarlatiniforme.
Acne.	Seborrhea oleosa.
Toxic erythema.	Duhring's disease.
Dermatitis herpetiforma.	Herpes.
Furunculosis.	Xanthoma.
Carbuncle (some cases).	Acne group.
Lichen planus.	Neurodermatosis.
Folliculosis sycosis.	Acanthosis nigricans.
Seborrhea (all cases).	Pruritus (with lichenization).
Seborrhea (some cases).	Melanoderma.
Eczema infantile.	

"Practically all skin diseases."

"The more the attention to the intestine, the better the dermatologist's results."

"No connection at all."

UROLOGY.

Pyelitis.	Proctitis.
Pyelitis (all types of infections).	Colitis.
Ureteral retention states (stric- tures also).	Pyelonephritis.
Chronic constipation.	Irritable bladder.
Vesiculitis.	Suppurative nephritis.
Varicocele.	Renal stone.
	Nephritis.

UROLOGY (*Continued*).

Prostatic obstruction (surgical).	Omentitis.
Prostatitis.	Cystitis.
Renal tuberculosis.	Cystitis fibrosa.
Constipation, spastic and atonic.	Non-septic metritis (female).
Hemorrhoids.	Urinary infection in general.
Diverticulosis (descending colon and sigmoid).	

"It's interesting; I don't know."

"Tremendously important."

"Of far more importance than urologists realize."

GYNECOLOGY.

Constipation.	Postoperative ileus.
Colitis.	Gastroduodenal ulcer.
Appendicitis.	Salpingitis (mixed infections).
Sigmoid flexure conditions.	Pelvic adhesions (otherwise no cause).
Rectal conditions.	Varicocele (in pampiniform from plexus of veins).
Adherent ileum.	Hyperplasia of endometrium.
Diverticulitis.	Cystitis.
Postoperative obstruction (some cases).	Pyelitis.
Intestinal and peritoneal tuberculosis.	

"Any and all of the disorders."

"Only contributory, of medical significance only, thus coincidental rather than etiologic."

"Not at all important."

PEDIATRICS.

Celiac disease.
 Hirschsprung's disease.
 Dysentery, bacterial.
 Intestinal infantilism.
 Rickets.
 Toxemia, high proteolytic flora.
 Toxemia, high acid flora (diarrhea, mucus disease).
 Chronic biliary dysentery.
 Sprue.
 Anemia.
 Chronic rheumatic conditions.
 Chronic malnutrition.
 Some forms of cough.
 Headaches.
 Anorexia.
 Eczema.

PEDIATRICS (*Continued*).

Nervous manifestations.

Constipation.

Mucous colitis.

Acute and chronic diseases of bacterial origin	{	typhoid.
		paratyphoid.
		dysentery.
		irregular para types.

Chronic intestinal indigestion.

Chronic dyspepsia.

"About all there is in pediatrics is disorders of digestion."

"Every condition in health or disease during the years of rapid growth."

"Too numerous to record."

Since from the above the deductions are easy, I will leave the reader to draw his own conclusions.

CHAPTER XV.

THE CLINIC.

GENERAL CONSIDERATIONS.

To compile and classify five thousand cases of intestinal toxemia in causes and effects, in etiologies and results, is practically an impossible task. There are many cogent reasons for this. Intestinal toxemia in etiology is rarely a matter of straight infections but usually mixed, and when mixed, it is difficult to declare accurately the importance of this or that organism singly, or this or that organism in the symbiotic make-up. Organisms of similar type biologies and definite biological forms are different in corporeal results, and in almost as many instances again, different individuals react variously to an identical organism. An infection being present, it depends not only upon the generic type as to what occurs or does not occur from its presence, but far more on the tissue, chemical and physical facts surrounding it, and beyond these are the still greater factors that are bound up in general and local immunity. The latter are many (and there are no ways at present to judge exactly what these are), and their powers or deficiencies keep the etiology itself in a maze.

Present-day biologic medicine has a great gap between the infecting organism on the one hand and what this organism can and does do in one instance, and does not do at all in the next. These are as much if not more important a part of the etiology as the organism itself. There is no doubt (seen notably, for instance, in streptococcic infections and existent also in many others) that different organisms of the same general types (cousins in a sense) can cause the same corporeal results. The unknown factors of more or less general and specific susceptibilities to types operate constantly in different persons and we have no worth-while means at present of classing the individual to even the general types of organisms, not to mention an identical species of one of the types.

Intestinal toxemia is a protean disorder encompassing the greatest of breadths in medicine and operating causatively and therefore therapeutically through the disorders and diseases in all of the departments of medicine. The matter is not only an infection on the one hand and a result on the other, but in the very essence of the etiology are many factors between the two, and these, in addition to anatomical, chemical and physical, are those of immunity, virulence of the infection, age, foods, length of time of the infection, routine of life, etc., the external environmental factors under which the person was, is set or continues, matters of elimination, oxidation, etc. Manifestly then to enumerate on the present-day basis of medicine of an infection cause and result, proper as it is in many disorders, is as valueless as it is impossible in the subject at hand.

To undertake to include in the report of cases the infection or infections found present, and the different biological methods of treatment with varying shifts in each case would mean the adding of an almost interminable amount of data which would only serve to confuse the reader. By it an already difficult subject would be rendered so difficult that no one could get anywhere with it, excepting into greater confusion. In the presentation of entities where it is possible to state the more numerous findings of definite infections, or those of type groupings, these will be made. But it must ever be remembered that the same organisms can cause to develop markedly different clinical disorders and diseases. The author presented for simple perusal of the numbers opposite the biologic treatments those which had been employed. Identifying the infection or infection make-up from stool examinations, one needs then but to resort to the tables of vaccines, subvaccines, antigens, antagonisms and sera, to accomplish quick direction of the biological methods that were employed in the author's cases. (I desire here to reiterate the importance of frequent re-examinations for guidance in proper and complete handling.)

To simplify the presentation of the vast amount of clinical material covered in the work, to make it more concrete to the reader, clinical entity terms in specialty groupings will be used as headings, and the context connected with each one will be

briefly given. No descriptions of disease or conditions will be presented. The especially interesting, or striking results (and there have been many) will be omitted. After several attempts of different sorts this seems to be the wisest method of presentation of the clinical side of this subject, even though it appears to be rather chopped and lacking in the value of continuity.

SYMPTOMS OF CLINICAL ENTITIES VERSUS COMPLAINTS OF PATIENTS.

Every physician is aware that the complaints of patients are the same in type and character occurring in simple functional as well as in obstinate organic disorders. There is a generalization about symptoms in which similar ones, or groups, may be significant or diagnostic of a disease entity, or they occur and no definite disease can be diagnosed or even suspected. To men of the highest abilities of clinical acumen patients will complain of the presence of certain symptoms and an explanation for their presence cannot be made on the basis of disease entity. The diagnosis of a well-established disease is not especially difficult when the symptoms and findings on examination fit in logical combinations. The rule here, however, is that the disease is well established, in which most times the interpretations of several physicians in attendance on the case could agree easily, or with only a small amount of question or debate. Well-established disease, though, whichever one it may be, is represented only in the smaller number of persons who complain and come under medical attention for symptoms of distress.

Of this larger number of all patients, three factors operate on the part of physicians of the past and present day. Firstly, if the physician believes that a definite disease entity is not present he minimizes its aspects, pays but little serious attention to the patient's complaints and dispatches the whole affair as a minor one to be gotten through with as quickly as possible. Secondly, there is the type of medical attendant in whom a cancer, for instance, must present itself as a large palpable mass before he can or does make a definite diagnosis, and there are many such in the profession in my experience.

There are all grades from this type to the somewhat opposite represented in the person who, if an x-ray examination is negative, finds there is nothing surgically wrong, and there being nothing surgical found, nothing at all is wrong, who accepts negative laboratory tests as definitely of negative diagnostic value, and finally the ones who depend more upon apparatus and such kinds of examinations rather than upon clinical ability in which the apparatus examinations would be mostly or considerably accessory, additional or confirmatory. Added to these are the deficient clinical types who do not use routine accessory means to help in diagnosis. Thirdly is the type who, if he cannot explain symptoms on the basis of disease, considers the case as neurological, endocrinologic, allergic, etc., and exercises an interest seriously in these connections.

The Complex Human Body.—The human body is a most complex affair in which endless disturbances or departures from the normal can and do occur. Many times a pathology is present to account for the complaints of persons but a pathology not completely enough advanced for diagnosis of disease entity. Such patients largely are dispatched into minor significance, really neglected, and the causes continuing to operate finally bring sufficient symptoms or findings to the fore, and generally, then, advanced definite disease exists and nothing in a curative way can be accomplished.

Much work is being done in the prophylaxis of such disorders as the exanthematous diseases, cancer control work, etc., but in the great field of primitive causes of the diseases of internal medicine which bring the vast majority of human beings into the grave before their time, any sort of worth-while work or attention is practically absent in the profession today. Cardiac, metabolic, arthritic, neurologic and other types of clinics are established or being set up and specialists are coming into existence everywhere and the material handled and receiving the total degrees of attention is commonly the result of long-standing neglected etiological causes. Numerous are the hospitals, operating rooms and the surgeons devoted to the results of neglected etiology. Almost totally the profession is

concerned with watching the fires, while there is no one concerned in fireproofing the human structure or in fire prevention.

Of course, there are causes that operate in disease production other than chronic intestinal states, but not only are these intestinal states the most numerous and potent of all in disease production, but of all the causes they are the poorest handled. This brings us back to the consideration of symptoms or complaints people bring to the physician. Excepting those people who treat themselves and those who have had no important or persistent enough symptoms to bring them under the attention of the physician, and the well-established entities, there is left the majority of patients who come under medical observation. They come to the physician complaining in some way or ways, and we classify their complaints under symptoms if disease is present. When the diagnosis is negative as to definite disease we should still recognize that a human being is not in total health who continues to complain of any of the symptoms mentioned in Chapter III, and here alphabetically arranged:

Abdominal distress and distentions.	Irritability.
Abdominal tenderness and pains.	Insomnia, fitful sleeping.
Abdominal colic and cramps.	Intestinal flatus.
Anemia.	Intolerance to acid foods.
Anorexia and fitful appetite.	Intolerance to starches, sugars or fats.
Arm pains.	Jaundice attacks.
Asthmatic attacks.	Joint pains, swellings and stiffness.
Bulimia.	Leg pains.
Back pains.	Leg weakness.
Cardiac palpitation and distress.	Lumbago.
Chest pains.	Mental depressions, melancholia.
Chills and chilly sensations.	Mucus from bowel or with stools.
Constipation.	Nausea attacks.
Corporeal itching (skin).	Neurasthenic symptoms.
Deafness.	Neurotic symptoms.
Debility, physical.	Obesity.
Diarrhea.	Pyrexia attacks.
Drowsiness, post prandial.	Rectal pains.
Dyspneic attacks.	Regurgitation (belching).
Epileptic attacks.	Skin disorders.
Fatigue.	Spinal pains and tenderness.
Functional eye conditions.	Stomach hyperacidity.
Halitosis.	Stomach hyperesthesia.
Head noises.	

Head pains.	Stomach pains and distress.
Headaches.	Stomach, weight and pressure.
Hiccoughs.	Subject to colds.
Hypertension.	Unconsciousness, attacks of.
Hypotension.	Vasomotor phenomena.
Indigestion (digestive disturbance)	Vertigo (dizziness).
gastric or intestinal.	Vomiting.
	Weight, loss of.

If most of the group complaints mentioned above are not of short-time duration, have existed for a length of time, and no reasons for their presence can be found, biologic study of the intestinal canal should be made for their possible explanation, and when abnormal biologies are found attention should be directed thereto. By routining this as a method of medical handling among physicians, specialists and surgeons, will be found the answers of much in prophylaxis, much in the explanation of failures of the past, and much in benefits of medical service heretofore not accomplished.

Matters pertaining to the diagnosis and treatment of intestinal toxemia should not eliminate any or all of the other things of significance in both diagnosis and treatment in the broad field of internal medicine and the specialties. The diagnosis of intestinal toxemia should not be made "a reason" for illnesses, without full and due consideration of all matters pertaining to medicine.

CHAPTER XVI.

INTERNAL MEDICINE.

Acidosis—Albuminuria—Anemias—Angioneurotic Edema—Arteriosclerosis—Asthma, Hay Fever—Diabetes Mellitus—Endocarditis—General Debility (Vital and Physical)—Hyperthyroid Goiter—Hyperthyroidism—Hypotension—Hypothyroidism—Myalgias—Myocarditis—Nephritis, Acute and Chronic—Obesity—Pancreatitis—Peritonitis—Pneumonitis—Rheumatism—Septicemia—Thyroiditis—Vasomotor Disturbances.

ACIDOSIS.

After all the studies and observations on this diagnosis which I have made, exactly what acidosis is as a clinical entity, I have never been able to understand clearly. If it represents a decrease in the amount of fixed bases in the blood and body by decrease of alkali reserve (a decrease in alkalinity), and if as causing this, starvation, anesthesia, pregnancy, certain infectious diseases (like rheumatic fever, typhoid, cholera), diabetes and cardiorenal disease can be thrown out of consideration, to me so-called acidosis is practically always a resulting state from a long-neglected intestinal toxemia and no other cause operates so significantly. What is stated here pertains as well to the cases of infants and children as adults. It includes also the opposite condition, alkalosis, the cause of which is from the same source, namely the intestines. According to the biological set-up in the intestines, in many instances the blood will give the findings of "acidosis" or "alkalosis" in varying degrees according to the individual and the intensity of the changed chemistry present.

In all of such instances, in the severe as well as in the more commonly met with mild cases, biological intestinal studies and handling are called for. When the acute symptoms have been met chemically and a cause is not manifest, the intestines as a source should always be thought of.

ALBUMINURIA.

In the so-called "functional," cyclic, orthostatic albuminurias, or albumosuria, always study the intestinal canal for their cause. In all of the "trace of albumin" urines, biologic study of the gut is also called for. Their numbers in my group are numerous, and the "simple albuminurias" number in the hundreds. Practically every one of them was an instance of intestinal toxemia and commonly the urines cleared after treatment. They often represent (when other causes for the presence of albumin can be excluded) some change in the glomeruli, the nature, extent and gravity of which cannot be estimated by urine examinations or be temporized with by leaving an underlying intestinal toxemia to continue. By allowing this, a considerable number in a few years will become definitely arterio-cardio-renal in disease, and then all opportunities for prophylaxis are lost. The life insurance companies are right today in generally refusing insurance to the albuminuric individual. While not a few of the whole will live out the ten or twenty years after the refusal, a significant number who continue a chronic intestinal toxemia do not.

ANEMIAS.

Secondary Anemia.—This form, distinguished from the acute type following hemorrhage, is commonly met with in practice. It represents the lower than normal blood in hemoglobin and sometimes erythrocytes found daily in clinical work. While it may be due to infections, repeated hemorrhages, and such intoxications as lead, mercury, etc., resorption of sulphur-containing toxic products from the bowel is the commonest of all causes. Such products seep into the blood stream from the lower part of the small and right half of the large intestine in a steady way, causing lytic effects upon the erythrocytes. Largely the biological findings in significance represent the proteolytic grouping of organisms of which there are many, and commonly inimical associations are present.

The handling is not only one of iron administration, or the correction of constipation and such measures, but more important is the correction of the intestinal state. In these, and in the primary or essential anemias, iron preparations or iron-bearing foods have rarely been used in my practice, the cases generally making perfect recoveries on correction of the intestinal condition.

Primary or Essential Anemia (Chlorosis).—This form is supposed to be of unknown origin, but it is due to intestinal toxemia. What is stated above fits perfectly in this more striking form of young girl anemia, excepting as a rule Bland's pills or some form of iron preparation is required for a short period in addition to attention to the intestinal infection present.

Pernicious and Borderline Types.—Up to the advent of blood transfusions, 18 cases of true pernicious anemia were treated by me by intestinal treatments. Although this disease was then held as "usually fatal," but three died of the disease and these were all advanced cases when first seen. The others recovered, with marked rises in red blood-cells and hemoglobin, which were sustained continuously over five years of time. The main treatments followed were biological and intestinal. After the advent of blood transfusions, which are so helpful, especially at the beginning of this disease, the results were quicker in clinical improvement but benefit from transfusions alone generally could not be depended upon. Of such nine were handled, all but one recovering (also an advanced case having had multiple transfusions when first seen).

Of late years "liver feedings" have limited the number of cases I now see, they being handled (excepting the very rapid cases) by the average physician successfully in symptomatic and continued ways in about three-quarters of the instances. A distinct infection of the *Clostridium putrificus* exists in all of the cases I have seen, this to me being the main etiologic cause of the disease, usually with symbiotic associations. "Liver feedings" merely supply the blood stream with the necessary blood constituents for the red blood-cells, the actual causes continuing to exist in the individual. For substantial results biologic treatments with liver diets are called for. What has

been stated applies to the borderline types as well. The toxic substance from this infection in some way brings on the achylic condition in the stomach and duodenum, and, either with this or because of this, causes interference with the running red cell production, a production necessary in health to meet the large breakdown of erythrocytes required for the carrying on of the functions of the body.

ANGIONEUROTIC EDEMA.

This disorder has been erroneously classed in the neurological disorders where it does not belong. Nor again does it belong to skin disorders because of its relation to urticaria, the giant form, and Henoch's purpura, which are supposed to be the same in cause. The manifestation is undoubtedly vasomotor, but I seriously question that it is, as Quinke believed, a straight neurosis. Of these cases there have been 37, all had an intestinal state, and practically all got well and remained so for years afterward, or had only minor and short-lived attacks at long intervals of time. This condition is due to intestinal toxemia as its main operating cause, and should be handled along these lines.

There are a few of these cases that are food allergic, but in my experience they mostly are allergic because of the intestinal condition. In these if the bacteriology and chemistry of the bowel is rendered normal they generally are no longer allergic. Allergic studies must include bowel chemistry (bacteriology) for understanding and control, and not just the intake of this or that food substance.

ARTERIOSCLEROSIS.

This disease has several causes, among which are the chronic toxic conditions. There has been much to prove in the work of several that chronic bowel conditions with constant toxicity are potent causes. Work has been done on intestinal toxic products, namely the oxy-products, to prove that vessel wall changes are attributable to them. With this there are others, like the effects of toxemias from bacteria, single or in

associations, which are still more damaging. No doubt in several ways a chronic intestinal toxic state can and does cause the condition and should be thought of in addition to other causes, such as senility, syphilis, excessive eating, and the toxemias of lead, alcohol, etc.

The element that gout plays is often also an intestinal question. What is likewise significant is that in conditions like aortitis (non-syphilitic), aneurism, periarteritis nodosa, involvements of the cerebral vessels, and the many conditions in which definite vessel involvement is present, intestinal biologic causes are significant in their etiology.

Once vessels are definitely diseased and a bowel condition is present, it is a question of clinical nicety whether or not biological examinations and treatments should be undertaken. In the advanced form of the disease manifestly nothing worthwhile could be accomplished. The mischief is done, and only temporizing methods are in order. But the majority of instances show vessel changes in a pre-age sense, whether with hypertension or not, which should be studied and handled in biologic ways. The instances of benefit and practical non-advance of the condition over ten years' time number into the hundreds of instances. Not to note the presence of minor degrees but definite arteriosclerosis in the young person is as much a matter of poor medical handling as allowing a causative bowel condition, if correctable, to continue on its shortening way to the grave in these conditions.

Angina Pectoris.—What has been stated above rules here, since one-half of these cases are due to obliterating disease of the coronary vessels, about one-fourth to thrombus or embolus (due to arterial disease or systemic infection from intestinal organisms), and the last fourth to syphilis or aortitis, the latter of which is not always syphilitic. Once true angina is established, nothing greatly worth while can be accomplished by bowel treatments, but in the milder and false forms, immediate intestinal study and treatment are called for. There were about 100 of such cases in my practice, the vast majority doing well in heart symptoms and conditions.

ASTHMA, HAY FEVER.

Bronchial.—Asthma and hay-fever are today considered largely allergic in origin. While there are some distinctly allergic types they are the least numerous of the cases. Mostly a chronic bowel infection is either the cause or is bound up in the anaphylaxis, the ingested foods being but one part of the problem, and commonly no real or direct cause at all. The so-called "metabolic" and "bacterial" exciting agents are commonly general terms for toxic bowel states. The so-called asthma which "runs in families" is merely an expression of a hereditary susceptibility of tissues. The afflicted individual generally has an operating cause altogether his own. The reflex neurotic form of asthma must be watched for and excluded. What has been stated here applies in some of the cases of bronchiectasis and many of chronic bronchitis, the first being due more distinctly to bacterial bowel infection, the second indirectly of bacterial and toxic origin.

In hay-fever generally three factors figure in the production, and commonly when one leg of this trilogy is taken away, benefit ensues. They are the pollen of plants, an abnormal condition in the nose, and a chronic intestinal toxemia. Most often the latter has erroneously been designated as a "neurosis," which when present is largely secondary.

DIABETES MELLITUS.

As a rule when this is well established the treatment is dietetic and by insulin. But to each case of true diabetes there are potential hundreds, instances of higher than normal blood sugars, occasional glycosurias, and those secondary to gall-bladder disease. Higher than normal blood sugars are definite resulting states from overeating (especially of carbohydrates) and deficient general body oxidation. Generally, though, it is symptomatic of an intestinal toxemia, the infection being usually of the saccharo-butyric bacterial groups of organisms. This, when long continued, causes the pancreatic cell tissue to break down in its efforts for over-oxidation; pathological disease then ensues which is permanent and real

cure is then not accomplishable. All of the valuable prophylactic days now are gone, and, as with so many other diseases, we spend our time in handling the result instead of its prevention.

In the usual potential case the author's pancreatic test shows the pancreas as efficient, and working in high figures of enzyme content in its external secretion (and that means internal secretion as well). Practically all gall-bladder disease is secondary to an intestinal toxemia, and, when constant urine sugar ensues from a lymphangitis which has extended over into the pancreas, pancreatic destruction or dysfunction is present. In this indirect way diabetes becomes the end station of the trip which starts in the intestinal canal. Quite obviously when diabetes mellitus is once established no cure can be brought about by biological treatments, but in the hundreds of instances of hyperglycemias of varying degrees, with efficient pancreas, the results have been most worth while, both in benefiting the condition and also in the prevention of the incidence of diabetes mellitus.

ENDOCARDITIS.

With the exception of the endocarditis due to syphilis and gonococcic infections, every organism causing this disease, including rheumatism, is a denizen of the intestinal canal. Additional rare infections are found in the diphtheria bacillus and meningococcus. But in the vast majority of all cases of both the acute and chronic forms, the intestine is the source of the original infection and this includes the ulcerative type. This statement pertains to any of the valves affected. The time to treat an endocarditis is before it has occurred, because when it has occurred it commonly is too late. When it is well established and compensation exists, an occasional case may be extended along and quick fatal mischief prevented; but these cases are few, and when the heart is broken down in its compensation, it is practically never curable. The same is true in the ulcerative processes.

GENERAL DEBILITY (VITAL AND PHYSICAL).

There are many instances of this condition in clinical work, usually in lesser degrees, and no definite disease cause can be discerned. In the instance where no cause can be found or factors ascribed, a biological study of the intestinal canal should be made. Commonly the cause is found there, and if so and proper treatment is undertaken, the results are often striking.

HYPERTENSION.

Blood-pressures vary greatly in different individuals and in the same individual under varying conditions. To get a fair index of what in a person is his blood-pressure, the test should be taken at least three times a day, for several days' time and under different conditions, and an average made of all the readings. But leaving out definite arteriosclerosis, alcoholism, syphilis, advanced years, renal and myocardial disease, and the menopausal cases, there is still left a large group of what used to be called "essential hypertension," in which spasms or tightening states of the smaller arteries exists (usually quite constant when definitely established) in which toxins from the bowel are the etiologic cause. Under treatment it is almost the rule in these cases to note the blood-pressure fall 10 mm. of mercury on biologic administrations of vaccines, reaching levels of better than 120 to 130 for those under fifty and 140 to 150 in those over that in a few weeks' time and remaining at these figures permanently. When definite organic disease exists in the kidneys not much benefit in lowering the pressures can be accomplished. This is true to a lesser extent with the myocardium, and definitely less with the blood-vessels.

HYPERTHYROID GOITER. HYPERTHYROIDISM.

Exophthalmic goiter is the result in the majority of instances of a long-standing severe grade of intestinal toxemia. The family predisposition is not entirely the cause—it usually requires a bowel condition in addition. Occasionally, of course,

it follows acute or local infections. Also I do not agree that profound nervous disturbances, worry, fright, etc., are the total fundamental causes.

As to "disturbances in the vegetative nervous system," these are usually conditions resulting from the bowel. Where the case is advanced and of some time standing so that the condition is accompanied by marked pathology in the thyroid, heart, etc., biologic treatments are usually too late to accomplish much benefit and other types of medical and surgical treatments are required. Not a few of these cases, though, can still be checked and markedly improved by proper intestinal handling. The great interest in surgery in this connection has as a rule come about by the cases not being handled properly medically but allowed to drift into extreme states.

Hyperthyroidism.—If definite pathology in the thyroid and thymus glands can be logically excluded or considered at a minimum, the case is a medical problem, and but very rarely surgical. While other forms of treatment (rest, sedatives, iodine, etc.) are in order, study of focal infection in the bowel as well as elsewhere is called for in all instances, and even occasionally in the hypertensive menopausal woman proper biological handling is necessary. The results here have been strikingly successful and almost uniformly so.

HYPOTENSION.

There are types of people who are hypotensive naturally. They largely are the thin, neurotic and ptotic types. Many variations are found among these. The main cause for continued low blood-pressures, however, is intestinal toxemia by the depressing effects of toxemia on the glands of internal secretion, mainly the thyroid group. The effects of toxic states on glands causing hypotension are easily demonstrated in convalescence after severe infectious diseases, the systolic pressure reaching normal standards for the individual when the last vestiges of the toxemia have been removed. So it is caused by toxic bowel conditions, only here there is a steady, devitalizing effect on the nervous system, causing fatigue, etc.,

this running constantly on. The rule is, although there are many exceptions, that once an intestinal toxemia causing hypotension has lasted long enough, more or less permanent mischief has occurred in the glands, in which case restoration to what we believe are approximately normal standards of health and blood-pressures is not generally seen. Largely in this group the organism set-ups of the proteolytic toxemias figure, although mixed types are often met with. The older the individual with hypotension from bowel toxemia, the less the systolic blood-pressure will rise by treatment; the younger, the more often it is seen.

HYPOTHYROIDISM.

Distinct cretinism is a hereditary condition, but there are many apparently acquired forms of subfunction of lesser grades (mixtures of the hereditary and acquired), cases in which degrees of low function are seen. Leaving out of consideration the cases which follow surgery on the thyroid gland (usually performed for excess function or pathology), there are not a few under-thyroid people in whom the atrophied glands are due to intestinal toxemias. The vast majority of thyroid cases ensue from slow destructive effects on the thyroid parenchymatous tissue from chronic intestinal toxemia. In these, so-called Gull's disease, seen varying in degrees, the intestine also furnishes the main etiological factors. This is especially true with the lesser grades, and of these there are many. While there is benefit in treatment with the thyroid substances, biologic study of the intestines is required, because it is not uncommon to accomplish perfect results without gland therapy, and these then are inclined to be permanent.

MYALGIAS.

The exciting causes of myalgia (myositis or fibrositis) are usually exposure and trauma. This condition takes place in susceptible types of individuals in whom intestinal toxemia may or may not exist. Where, however, it occurs frequently from slight causes, the possibility of a focal infection should be

thought of, and this in the intestines always. This holds true in lumbago, ischemic lumbago, spinal arthritis, sacro-iliac relaxation, neuritis of the posterior nerve roots, torticollis, pleurodynia, cephalodynia, scapulodynia, omodynia, dorsodynia, pes planus, and tennis elbow of persistent types. In the acute case when the cause is plain and distinct and the course short, temporizing treatment may be engaged in. But where these conditions play prominently in the individual, careful analyses of the bowel condition and appropriate treatment are in order.

MYOCARDITIS.

Practically every case of chronic myocarditis, whether disease of the valves is present or not, whether chronic nephritis or arteriosclerosis is present or not, no matter what the status of hypertrophy, dilatation, or cardiac insufficiency, when the well-known causes can be ruled out or are not present, is a neglected case of intestinal toxemia. When distinct disease of the myocardium has occurred it generally is too late to bring about benefits from biologic handling, although occasionally it is helpful in worth-while ways. The histories of most of the cases I have seen have the possibilities of intestinal toxemia written plainly in the past story of the patient. This holds true in the majority of the cases of chronic heart, kidney and vascular disorders. The sooner the profession realizes when such cases are seen, that causes, generally intestinal, have been operating for years without wise attention, the sooner are we going to control a large part of the rapid advance of cardiac disease. Manifestly, when distinct myocarditis is present, the treatment is largely only symptomatic and of the general types.

NEPHRITIS, ACUTE AND CHRONIC.

Acute.—The commonest cause of acute nephritis is the toxic effect of an infection upon the kidneys. Intestinal toxemia is always an infection. Exposure, scarlet fever, etc., definite chemical toxic agents (mercury, turpentine, etc.), pregnancy, operations upon kidneys, extensive skin diseases (burns,

etc.), can be causes, but in many cases the etiology is none of these, but intestinal. Every instance of acute nephritis in the young or middle-aged without the evident causes calls for biological study of the bowel. Especially is this important because the cause may continue to operate and chronic nephritis eventually ensue. There have been 32 cases of this in my practice, all intestinal in origin.

Chronic.—Some of these cases, of course, follow an acute course. But it is agreed that continued bacterial septicemia secondary to focal infection is the most important cause, and these cases are slow and insidious. Since the intestinal canal is the most important and commonest of focal infection areas, when fevers, malaria, alcohol, syphilis, or tuberculosis can be eliminated, intestinal studies are in order. This holds true with the chronic parenchymatous as well as the arteriosclerotic types of the disease, and the latter all the way through the significances of hypertension, myocardial insufficiency, and even when uremia in its varying grades had been or is present. Every case of chronic Bright's disease should be thought of and studied from intestinal standpoints, and the vast majority will be found to require biologic bowel handling.

It must be apparent, though, that when advanced and permanent destruction of the kidney tissues has taken place, one is left only a symptomatic policy in treatments. Receiving the case early enough, and the majority are in this grouping, some of the most remarkable results can be accomplished by proper intestinal toxemia handling.

OBESITY.

The profession does not seem to be aware that obesity in many instances is a symptom of intestinal toxemia. Leaving out the hereditary, excess eating, menopause, post-pregnancy, and the pituitary cases, there are a large number in whom excess fat deposit occurs from deficient metabolic causes, secondary to gut infections, usually of the saccharo-butyric group of infections. Of these there are some twenty or more bacteria, straight, but usually in combined infections, responsible

for the sudden addition of weight or the difficulty of losing it on diets and proper treatments for obesity. The obesity here is merely a symptom, a train of associated events of ill health often accompanying or shortly following its presence. It is interesting to observe a drop in weight (independent of regulation of foods) when an intestinal infection is being controlled, and not uncommonly the drop in weight remains stationary irrespective of dieting in substantial ways.

PANCREATITIS.

Interlobular and interacinar pancreatitis is a far more common condition and the producer of digestive symptoms and general states of ill health than is supposed. Leaving out the instances of malignancy, syphilis, or tuberculosis (all of which generally affect the head of the organ and therefore the most important part), there remains the majority of the instances of this condition. The author's pancreatic test proves this clinically as well as the reports of autopsies and those from surgeons. Chronic pancreatitis is caused especially by intestinal toxemia, either directly from toxic effect on the acinous tissue or again secondary to gall-bladder infections, these being due to intestinal organisms. Whether with glycosuria or diabetes, the main interests in the condition are the prophylaxis connected with diabetes and the betterment of intestinal digestive disorders and general states of ill health and subnutrition. The pancreas, like the heart and liver, has a remarkable reserve and recuperative power. Time and time again the reestablishment of the gland's function when an intestinal toxemia is removed has been noted.

PERITONITIS.

Primary acute peritonitis is generally due to the pneumococcus or one of the streptococci which reach the peritoneum, not by blood stream or lymphatics as is generally supposed, but mostly by direct invasion from the interior of the gut. Anything that lowers the general vital resistance, like nephritis, gout, or arteriosclerosis, also lowers the protective

powers of the peritoneum—hence the frequency of the condition as a terminal infection. The peritonitis of septicemia and pyemia, almost invariably the result of a local process, is in the original local process generally intestinal in origin. Every organism found in any of the forms of peritonitis is that of an intestinal type (excepting the tubercle bacillus). The same significance holds good in subphrenic peritonitis, and in the chronic forms of peritonitis of the local adhesive, diffuse adhesive, proliferative and chronic hemorrhagic forms. One needs but to state the rule of the intestinal origin of these infections—the infection being first in the lumen of the gut, then in its walls, and finally outside—for significance to be manifest. This is the main point, and according to the case it has importance.

PNEUMONITIS.

The pneumococcus is a frequent denizen of the human intestinal canal, often found in non-toxic states, and its kin, the *Streptococcus pyogenes*, is commonly associated with it. Its mode of invasion of the lungs has not received the study it deserves. The supposition holds that it enters by way of inhaled air or insufflation from the fauces, where it may often be found in health. It is probable that a common mode of invasion into the lungs is by way of the lymphatics of the post nares, or throat, by directly descending, or indirectly by blood stream, finally lodging in the fine capillaries of the lung tissue. Since all bacteria have the faculty of traversing the gut walls, it is possible that invasion into the blood and lodgment in the lung capillaries may occur in this way. It is probable that in the aged and as a terminal pneumonia it often occurs in this latter way. The same holds true in bronchopneumonia, in which the staphylococcus, the pneumococcus and the *Streptococcus pyogenes* are the commonest of the infecting organisms. These points are merely mentioned in this connection as of interest in the bowel being an original source of infection which may require subsequent attention.

RHEUMATISM.

The infection here is one of several strains of the streptococcus and, in my opinion, is largely intestinal in its original source. The combination of arthritis, myocarditis and endocarditis with the general symptoms, strongly suggests that it is an infection. In recurring attacks of what is designated as acute or subacute rheumatic fever in children and adults, biologic studies of the intestinal canal should be engaged in as well as other sources of focal infection. Some of the best results in the prevention of attacks have been accomplished where immunity against streptococci could be accomplished. Even in the presence of the acute stage, intestinal studies should be made. Sometimes to save the heart from irreparable damage the correction of an intestinal state is most important.

SEPTICEMIA.

The above term is used to include a group of conditions clinically designated as sapremia, septicemia, cryptogenetic septicemia, pyemia, focal and terminal infections. Since these infections may originate from any local source, the bowel should always be thought of in the absence of local lesion, because most commonly they are due to the *Streptococcus pyogenes* and other strains, the *Proteus*, *Pseudomonas aeruginosa*, staphylococci, *Escherichia coli*, and pneumococci, all organisms of the intestinal canal. As a rule, one is not given much chance in the presence of alarming symptoms to conduct any intestinal studies or treatments. Occasionally, however, a subacute or more chronic case would be met with, in which instance it is always important that these studies be carried out, since there is danger of reinfection and the ultimate course and result may be dependent largely upon attention to the intestines. In 9 cases of the acute form, and 16 of the recovery form (all with positive blood cultures), the organisms present in the blood were also present in the intestinal canal and persisting in the latter when the blood infection had cleared.

THYROIDITIS.

When typhoid fever, smallpox, measles and mumps can be excluded as a cause, even though pneumonia or rheumatic fever is present, the intestine should be thought of as a source of the original infection. In these rather rare cases destruction of the gland may be considerably modified by this thought being kept in mind and attention given to the intestine.

VASOMOTOR DISTURBANCES.

The most prominent of these is Raynaud's disease, the arteriosclerotic form being included in significance under arteriosclerosis. The spasm in the arteries and veins in the majority of instances is caused by toxic products from the bowel affecting the ganglia and neurons of the sympathetic system. All such cases require intestinal study whether they are just local and transitory or the more severe forms. I have had no experience with erythromelalgia. Angioneurotic edema is described in the fore part of this chapter. One case of facial hemiatrophy was due to a chronic streptococcus infection of the bowel. There are many transitory and milder forms of vasomotor disturbance, more functional types, all of which should have biological study.

CHAPTER XVII.

GASTRO-ENTEROLOGY.

Abdominal Adhesions—Achyilia Gastrica—Appendicitis—Atonic Colon—Chronic Constipation—Colitis—Colitis, Ulcerative—Diverticulosis and Diverticulitis—Erosions of the Stomach—Gall-bladder Infections—Gastric Disturbances—Gastritis, Chronic—Gastro-enteroptosis—Gastro-intestinal Atrophy—Hemorrhoids—Hyperacidity, Hyperchlorhydria—Ileocecal Incompetency—Indicanuria—Intestinal Stasis—Neurotic Abdomen—Omentitis—Proctitis—Pruritus (Anal)—Pyloric Obstruction—Pylorospasm—Redundant Colon—Spastic Anal Sphincter—Spastic Colon (Constipation)—Spastic Recto-sigmoid Apparatus—Ulcer (Gastric and Duodenal).

ABDOMINAL ADHESIONS.

Leaving out of consideration the embryonic type of adhesions, as well as those caused by surgery, injury and otherwise, the commonest cause of adhesion production in the abdomen is an intestinal toxemia in which the organisms have traversed the walls of the intestinal canal, causing a plastic type of peritonitis; or a plastic type of peritonitis occurs to protect the peritoneum and body from invasion, and this, touching a proximal peritoneal surface, causes the same process to occur, the two uniting with ultimate band formation. Such adhesions may occur anywhere along the course of the intestine, but most often are found in the lower portion of the small intestine and the colon. The condition known as "adherent ileum" and all of the instances of pericolonic adhesions are due to intestinal infections. Since abdominal adhesions is a diagnosis so often made and a condition so commonly found at operation, it is a wonder to me that there is so much interest in it and none at all in the study of the diagnosis, or the removal of its cause. I have seen colons, adherent practically throughout the entire length within the abdomen, where the best diagnosis made by the best of men has been "colonic adhesions," and usually the only thing decreed to be done was a surgical procedure.

I wish here to go on record, that while knowing this, in the vast majority of instances of well-established adhesions, especially when they are obstructive, but little can be done in the way of benefit (except surgery in cases of obstruction and gut anchoring), I am firm in the belief, which was critically arrived at, that in not a few cases, some even in troublesome types of adhesions, the removal of an intestinal toxemia has caused a betterment to such an extent that it was not possible to demonstrate adhesions afterward. That means that it is often possible, when an intestinal toxemia can be corrected, to find a disappearance of the dynamic factors of adhesions in the abdomen. With removal of cause, it is often possible to bring relief in the symptoms due to congestion and hyperesthesia (sensitiveness). The presence of unexplained cause of adhesions sanctions biological attention.

ACHYLIA GASTRICA.

In the first edition of my work on Diseases of the Stomach published in 1910, and maintained regularly up to the present sixth edition, leaving out of consideration the short-term neurological cases, fear from extraction of test meals, and those who had been born with a non-acid stomach, I held that the condition clinically known as achylia gastrica was mostly a secretory diagnosis made in the late stages of a chronic gastritis. More recently, Faber, as the result of study of the gastric mucosa in these cases, substantiated by many abroad, presents as the summary the following: "We arrive at the conclusion then that the very common and very important disease which we usually call achylia gastrica has nothing particularly remarkable in its etiology and pathogenesis which marks it out from diseases of other organs. It is due to disease of the gastric mucous membrane with its extensive glandular apparatus. When it is excited through nervous channels it can only persist for a short time, but apart from this it is due to the same causes which we know produce disease in other organs—direct irritation or hematogenous intoxication by bacterial toxin or autotoxin. The natural term for the disease is gastritis by which it is to be understood. Not the old con-

ception of gastric catarrh but a disease of the glandular parenchyma of inflammatory nature."

Personally I have yet to see a case of true achylia gastrica in which a chronic intestinal toxemia does not exist, and the rule is that the toxic state in the bowel is a pronounced one. When a condition exists like sprue, gout, arthritis deformans, any of the infectious diseases like tuberculosis, or parasitic diseases like uncinaria or hookworm disease, in states of chronic passive congestion such as those due to cardiac and renal conditions, achylia usually exists, and as a rule this achylia is due to a granular degeneration of the cells of the gastric mucosa caused by the toxemia of these diseases or conditions. It is almost a truism, when a non-acid stomach cannot be accounted for on some clinical basis, that the persistency of a non-acid test meal or those with very low acid trends are suggestive of intestinal toxemia.

Here and there, in the last twenty-five years, when some degree of acid-secreting ability is still present, it has been found that when the intestinal toxemia could be markedly benefited, the stomach is able to secrete a gastric juice higher in quantity, HCl content, and enzymes. As a rule, however, instances of achylia gastrica have been neglected so far as the intestinal canal is concerned, and when the destruction in the gastric mucosa is advanced no restoration or reconstruction is possible. The infections here belong mostly to the aërobic organisms of which there are many types capable of producing the condition.

APPENDICITIS.

The appendix is located at the site of the greatest bacterial activity that goes on in the intestinal canal. The organisms here are practically all of them viable, the lytic and disintegrating changes and antagonistic activities of the transverse and left colon not having had time to operate upon them. If the appendix is healthy (and that means the intestinal mucosa, so that no extension through it can occur) it usually functions well so it can empty and drain its interior. But if right-sided changes in the mucosa have occurred (and they are very fre-

quent), or the appendix is subcecal so it cannot empty readily, or is anchored by a fold of the peritoneum (and thus fixed), it cannot drain easily. Its contents will then inspissate, with resulting infection of the lymphatic appendix walls, or if the virulence of too long retained organisms is high, infection occurs. In both of these instances an infection of the intestinal contents is always present, and the acute, subacute and the chronic forms of appendicitis are indicative of intestinal toxemia.

In all instances of an acute appendicitis a biological study of the gut should be made, whether the appendix is removed or not, the same being necessary in the chronic involvements. In the latter case if this were done not so many appendices would be removed to cure right-sided pains and other clinical make-ups in which no benefit to the patients from appendectomy takes place. One cannot cure an intestinal toxemia by the removal of an appendix, and cannot cure a right-sided pathology in the mucosa by it either. One needs but to study the constantly increasing army of people who have been appendectomized for chronic appendicitis uselessly, and who are simply victims of poor diagnostic work as well as examples of short-sighted therapeutic work.

Argument has been presented that the removal of an appendix will cure or alleviate an intestinal toxemia, that an appendix drains infection into a bowel and so forth. All facts and reasons are directly opposite to these. The removal of an appendix to cure intestinal toxemia is about as reasonable and sensible as the removal of a dog's tail to change the color of the hair on the back of the animal. The presence of a diseased or infected appendix is evidence enough for the need of biologic study of the intestine to be made, because the following array of organisms from infected appendices—from the work of several investigators as compiled and presented in the text-book of Dr. Kelly¹—demonstrates the prevalence of intestinal organisms as the cause of infections: *Escherichia coli*, *B. aërogenes*, *Micrococcus pneumoniae*, *Micrococcus au-*

¹ Kelly: *Appendicitis and Diseases of the Vermiform Appendix*, 2d Ed., p. 47.

reus, *Streptococcus pyogenes*, *B. subtilis*, *B. lacticus*, *Pseudomonas aëruginea*, *B. foetidus liquefaciens*, *Diplococcus intestinalis*, *Clostridium tetani*, *Bacteroides fragilis*, *Bacteroides ramosus*, *Clostridium welchii*, *B. fusiformis*, *B. forcosus*, *Proteus vulgaris*, *Aërobacter aërogenes*.

To the above the author could add twice as many more different organisms from appendices removed from patients in his practice, these being identified through better methods of cultural growth and study. But the array above is sufficient to prove conclusively that the appendix is always infected from the intestinal contents and it depends mostly upon the symbiosis of the above with other of the intestinal organisms (and this is a most important matter in the understanding, diagnosis and proper treatment of an intestinal toxemia) to understand practically all of the appendix infections.

Chronic Appendicitis.—This condition, rather questionable in significance in medicine, in my opinion is a resulting state of a past appendicular infection in which the appendix by replacement tissue has protected itself against acute and subacute inflammation. There is a thought in medicine that acute appendicitis is merely a flare-up of a chronic appendicitis. This may be true in some instances, but what is more definitely sure is that in chronic appendicular disease infection had been in the appendix for a long time. I am of the opinion in cases of definite chronic appendicitis where there are no direct symptoms and in which appendicular dyspepsia can be ruled out, that surgical procedure, in most instances, is after nature has cured and protected the organ. Even more than in acute appendicitis after surgery has been done, to allow the chronic case to be dismissed without further study and attention, is limited handling. Every instance of acute, subacute, or chronic appendicitis, whether the organ is removed or not, requires study of the biology of the intestinal canal.

ATONIC COLON.

The commonest pathology in the human being as proven by autopsy is small hemorrhagic lesions of the colon accompanied with destruction of the follicles and degeneration and

necrosis in the mucosa, mainly in the glands and lymph nodes, commonly with definite desquamation of glandular tissue. In the first edition of my text-book on Diseases of the Intestine this point was brought out with this important addition, namely, that in these instances of mucous membrane change from long-standing intestinal toxemias, the cells of the Meissner and Auerbach plexuses are shrunken, stippled with a fine granulation, the nuclei and neurons showing distinct change from normal. Following the sympathetic centrally from the plexuses of Meissner and Auerbach, including the ganglionic cells on the way, and even as far back as the large sympathetic plexuses in the back of the abdomen, degeneration of essential elements of this system often takes place.

It must therefore be evident that when this portion of the sympathetic system has been more or less cut off, dilatation of the organ to which these fibers are distributed takes place. The process then becomes a vicious circle beginning with infection within the bowel, changes in the mucosa from toxic and lytic products of bacteria, and changes in the ganglia and neurons of the sympathetic plexuses from these same causes. There then occurs dilatation and deficiency in the dynamics of the bowel with further toxemia and further pathology. Therefore, in all instances of atonic colon, mild degrees of mesenteric ileus, incompetent ileocecal valve, mobile and dilated cecum, idiopathic dilatation (Hirschsprung's disease), and elongated colon, intestinal toxemia should be suspected and biologic examinations and treatments engaged in. Some of the best results found in the biologic handling of intestinal toxemia are in these conditions, and this also includes dilatation of the stomach, non-obstructive in type. The infections here are usually of the mixed or associated groupings, commonly those producing definite proteolytic effects from the food pabulum in the digestive canal.

CHRONIC CONSTIPATION.

There are many causes of chronic constipation, such as improper eating, ignorance, laziness, false modesty, purgation habits, flexure defects and malposition, senile and exhaustion

states, strictures, hypertrophied O'Beirne's sphincters, hypertrophied Houston's valves, hypertrophied levator ani muscles, etc. But the commonest of all causes of chronic constipation are the atrophic changes which occur in the mucous membrane, usually accompanied by atonic conditions in the gut wall secondary to long-standing intestinal toxemia. It is interesting, and the cases here have been many and the experience vast, to note that when it is possible to make an accurate diagnosis and accomplish good quality of biologic handling, without diet, medication, constipation food additions, or anything of like character, the mucous membrane becomes normal in appearance and the constipation terminated. This dry colitis, or colitis sicca, is an exceedingly common condition, and when the intestinal toxemia has continued for any length of time, the constipation itself is one of the most vicious causes of further increase in the intestinal toxemia and this again causes more constipation. The infections that can cause this are numerous and the mixtures of these infectious organisms still more so.

COLITIS.

Attention here is directed mainly to the chronic form, variously designated as mucous colitis, mucous colic, membranous colitis, etc. Thought, attention and reams and reams of paper have been devoted to chronic colitis. Many of the best men of medicine believe the condition to be largely a neurological affair, requiring neurological types of treatment, such as mental correction, rest, diet, vacations, etc. I wish here to go definitely on record that while more or less unstable types of people nervously are more prone to this disorder, the condition is fundamentally not only a neurological one, but largely a secondary condition to intestinal toxemia.

This viewpoint is arrived at after the treatment of over one thousand of these cases in which it was a far more common experience that the so-called neurological condition was secondary to the intestinal, and not the primary cause. To treat mucous colitis in all of its shades and conditions as is being done in medicine today is distinctly short-sighted and

largely means the continuation of the complaints and troubles. Most numerous these cases pass along from one internist or gastro-enterologist, or neurologist, to another, being bucked-up for the time being by the treatments now in vogue, and then they start again in a new medical direction. Until the profession realizes how much a chronic colitis is a secondary condition to intestinal toxemia, many of these cases will continue along uncured.

As a rule the instability in the vegetative nervous system is induced from the bowel state. The brightest parts of my work have been in the handling of these people biologically, up and around, with no rest cures, medication, or other things, and seeing the remarkable results accomplished in the bowel mucosa, general condition, and status of nervous system, these results generally being permanent. It is my opinion that the mucus with the stools, or passed without feces, is Nature's way of protecting the bowel from the toxemia. Then why wash out the colon and the use of various medicaments in the solution, like sodium bicarbonate, hydrastis and ichthyol, just because mucus is present? The mucus is only a resulting symptom and is not a substance to attack because it is present. The same is true of the spasm and spastic states and the employment of various symptomatic remedies therefor. The majority of these are instances of mixed infections of which there are many different groups and kinds.

COLITIS, ULCERATIVE.

Ulcerative colitis is not any more due to the Gram-positive *Diplococcus intestinalis* than pulmonary tuberculosis is due to organisms mixed with the tubercle bacillus, such as streptococci, staphylococci, etc. This organism (erroneously designated the "Bargen bacillus") was first described by Escherich in 1886 as the *Micrococcus ovalis* and subsequently by Conradi, Kurpjuweit, Moro and Murath as the Gram-positive *Diplococcus* or the *Diplococcus intestinalis*. The designating of this organism by the profession as the "Bargen bacillus" is a good index of the lack of knowledge in the profession of biological matters and of the intestine in particular. Bargen

drew attention to it as the exciting cause of ulcerative colitis, which it is not, being only and always an associated or more correctly a subsequent infection. Before Bargen spread literature on it in intestinal work, the organism had long been known (because it is a denizen of the normal intestinal canal of man), and it is not even a bacillus but a coccus instead. Since there is so much interest in the biology of this subject now, the infective causes, of ulcerative colitis in straight and associated infections as found by me were the following:

Streptococci (any of the 16 strains of the Holman classification).

Staphylococci (mostly the albus).

Eberthella (19 strains).

Salmonella (9 strains).

Escherichia (9 strains).

Alcaligenes (5 strains).

Aërobacter (1 strain).

Clostridia (9 strains).

Bacteroides (11 strains).

There are three main causes of ulcerative colitis: first, infections with the above organisms; second, lack of resistance, both local in the bowel and general in the body against these infections, largely, but not entirely, because of the youth of the patient; and third, a food deficiency factor which we know little about but probably is bound up in vitamins or inimical food mixtures. Quite obviously, then, the diet should be ample and generous, resistance built up in every possible way, and the infection controlled by biologic means according to the infection. In these cases, blood transfusions, liver diets, blocking the lymphatic structures (colonic, pericolic and mesentery) with intravenous mercurochrome to control reinfections were found important and always in order, as is occasionally a cecostomy for irrigating purposes from above the infection downward rather than by the anal route (which carries the infection upward), or an iliac implantation in the rarest of instances.

Of such cases with definite ulceration there have now been 104 biologically handled. One had an iliac implantation, 19 were cecostomized, and all were treated entirely medically and

biologically with but one death, and in all other instances there was a cure. Often the latter took several years of time to accomplish, but in the majority this was accomplished inside of two years. Only those that have remained well for the space of three years are recorded.

DIVERTICULOSIS AND DIVERTICULITIS.

In my opinion diverticula, usually of the colon—although they may occur anywhere, even as high as the duodenum—have had presented about every sort of etiology to explain their occurrence but the one of chronic intestinal toxemia, which is the commonest of all causes, in fact perhaps the main one. With an intestinal toxemia present (especially when there is retention in the colon and constipation, which I explained before is usually due to toxic reasons) in some individuals where there is localized weakening in the muscularis or at the “vessel holes,” diverticula may occur as they do in about five per cent of all individuals who are toxic. Of course where a carcinoma has developed on the diverticulum, definite obstruction usually exists, or in a diverticulitis with the formation of abscess, the cases primarily are only of surgical interest. But even in these, and in the far more common ones of the self-limiting diverticulitis and peridiverticulitis, or where just diverticula are present, to do no more than to make a diagnosis and to establish some type of treatment of short duration is definitely below what proper handling should be in the majority of instances.

EROSIONS OF THE STOMACH.

See Section on Chronic Gastritis.

GALL-BLADDER INFECTIONS.

The commonest cause of gall-bladder infections and gall-bladder pathologies is the presence of an intestinal toxemia and infection in the bowel, the bacteria having gained entrance into the portal veins, traversing the liver, and being eliminated in the biliary radicals from which bile collected in the gall-

bladder infects the sac wall. There have never been bacteria isolated from an infected gall-bladder or bile therefrom in which the infective organism was not a denizen of the intestinal canal in the same individual.

To routinely do a cholecystotomy on a case for removal of stone or drainage of infection of the gall-bladder should be discontinued, the only surgery indicated when definite pathology or infection exists being removal of the sac. The removal of the sac, however, does not cure the intestinal toxemia, and the surgeon who thinks that after its removal the case is closed and nothing further need be done does not meet the requirements in by far the largest number of cases. In addition to this the medical man who treats cases of chronic cholecystitis with medical bile drainages, medications, diet, etc., neglecting the most important cause—a constant reinfection of these structures from the intestinal canal—is quite as short-sighted in proper management. It is an interesting finding, seen in numerous instances where a chronic cholecystitis was present, by the correction of an intestinal toxemia (with nothing definite or centralized being done to the gall-bladder) to have the patient become entirely free from gall-bladder symptoms and remain that way permanently.

The pathological studies of the gall-bladder wall, the bile contained in removed gall-bladders, and the specimens obtained from biliary drainages show a high incidence of positive cultures of bacteria in various pathologies of the gall-bladder. So steadily present are bacteria that it may be questioned that pathology of the gall-bladder can occur without an infection being present and that an infection is always importantly bound up as the cause of gall-bladder pathology.

Nobody seems to have been concerned as to where these organisms originate, all the attention having been directed to medical and surgical treatment of only the gall-bladder itself when pathology or infection is present. The important question now is where do these infections come from, and if they also are present in a locality removed from the gall-bladder, have they significance so far as the gall-bladder and other conditions are concerned?

In one hundred examinations of bile (in other than hydrops of the gall-bladder, in which the contained fluid is usually sterile) the following represents the bacteria met with. These were: *Escherichia coli* infections, non-hemolytic streptococci infections, hemolytic streptococci infections, *Staphylococcus aureus*, *Clostridium putrificus*, *Staphylococcus albus*, *B. pyocyaneus*, Gram-positive micrococci, *Eberthella typhi*, *Pasteurella* Sp., *Eberthella metadysentericus* A, *Salmonella para-aërtrycke*, *Salmonella schotmülleri* A, *Salmonella schotmülleri* B, *Alcaligenes fæcalis*, *Eberthella dispar*, pneumococcus, *Escherichia neapolitana*, *Escherichia pseudocoloides*, *B. prodigiosus*, *Streptococcus fæcalis*, *Escherichia colotropicalis*, and others less frequently found and some difficult of identification, but in all probability intestinal in origin.

These are all denizens of the intestinal canal. Exception may be made in the non- and hemolytic streptococci, but if one has done enough work with stools one soon learns that they also are common intestinal organisms and rather easily cultivated from stools and commonly from those in which they would not be expected to be present in the numbers often met with. All or any of them gain the biliary passages by passing from the portal blood into the bile, the liver happening not to be sufficiently bactericidal to destroy them. A point here of interest is that the bile in these instances, because of it being poured into the intestine, acts as an eliminator of bacteria which have gained the liver. The hepatic veins are a small absorbing area compared to the larger bile-secreting area, and Nature logically would prefer to send the organism to the biliary radicals rather than through the more dangerous route into the general blood stream.

No doubt certain functional deficiencies of the liver (regarding which practically nothing is known, and yet which must occur as in all other organs of the body) and non-diagnosable states of liver pathology, would help in bacteria coming through in the bile. When eliminated in the bile, tissues over which the bile runs and collects may therefore become infected and this no doubt accounts for the large incidence of gall-bladder pathologies compared to others met with in the abdomen.

Another point here is that there are not a few infections of the intestine (in those of even early middle age) in which pathology in the gall-bladder may be assumed with accuracy in practically all instances. Briefly, as a class, they represent the infections met with in the grouping of the saccharo-butyric toxemias, although they occur with others. One must now strongly question all the activity and interest the profession has displayed these last years in debate on the medical and surgical diagnoses and treatments of chronic gall-bladder conditions.

For years interest and controversy have waged on matters pertaining to the so-called Meltzer-Lyon methods of diagnosis and treatment, vaccine treatments, etc., and were it not for the purgative effect of the magnesium sulphate solution instilled (and now added to by many with a transduodenal lavage), these cleaning out the intestinal canal from above downward, all would have been a waste of time, money and paper. Why? It must be perfectly manifest that one cannot cure an infection of the biliary passages by medical bile aspirations. No doubt a temporary benefit does come here and there. But where is the great virtue of it when the causative intestinal infection is left as a fire under it constantly reinfecting or, as is most often the case, keeping the infection up? There have been enough instances of infective gall-bladder cases treated by biological methods alone to quite convince me that the best form of treatment for such gall-bladder conditions is that of the fundamental trouble in the intestine.

And so, too, with surgery. If the gall-bladder is removed, local symptoms, direct and referred from that viscus, may be benefited, but it must be remembered that every instance of gall-bladder pathology and infection is secondary from the intestine and one needs but to prove the existence of such gall-bladder condition to also prove the presence of an intestinal infection, and no form of treatment, medical or surgical, can be altogether complete when attention is directed only to the gall-bladder state.

GASTRIC DISTURBANCES.

Intestinal toxemias are the commonest causes of the production of gastric hyperacidity, subacidity and anacidity, gastric hyperesthesia, gastric atony, gastric hyperperistalsis, and spasm. In some, the toxic products in the blood act directly upon the stomach structures, and in others they act reflexly. Books and articles are written on the above conditions, and no one seems to pay any attention to the commonest of all the causes. I have seen case after case, after they have been in the hands of conscientious men, in which powders, tonics, diets, etc., have been used for years without any permanent benefit. It surely should have been evident to them that the cause, or the causes, operating are not only local or have to do not only with food, habits, etc. The sooner gastro-enterologists enter into this line of interest the sooner will they accomplish success in the handling of many of the chronic or constantly complaining gastric cases.

GASTRITIS, CHRONIC.

What has already been stated in connection with achylia gastrica holds true with chronic gastritis. Leaving out of consideration the instances of chronic gastritis which occur from rapid eating, imperfect mastication, over-feeding, excessive drinking of acid fluids, intemperate use of alcohol, tea, coffee and tobacco, and those instances in which chronic gastritis occurs as a secondary disorder, the commonest causes of simple, chronic, hypertrophic, sclerosing and atrophic gastritis are intestinal in origin. Likewise not a few instances of recurring acute gastritis, all instances of the suppurative forms, the infectious, hemorrhagic and erosion types, require careful biologic study of the intestinal canal. Of course in acute suppurative gastritis there is no opportunity to do this, but in the other types the explanation of the presence of these conditions will be found largely in biologic errors in the lower levels of the digestive canal.

GASTRO-ENTEROPTOSIS.

It is most probable that in true gastro-enteroptosis, other than in the post-pregnant woman of the thin type, we are dealing largely with a congenital and hereditary condition. However, there is no doubt that intestinal toxemia plays a large part in the production of what may be termed the loss of balance in which symptoms are produced in these people. Up to the present time gastro-enterologists have handled such patients with abdominal support, diet, elevated bed, tonic, rest cure, etc., and they have accepted the return of these people after a few months' or a year's time as being logical in connection with ptosis. In at least half of the definite instances of ptosis, whatever the treatment, whether it be ambulatory or the bed type, to leave an intestinal toxemia means the reestablishment of the symptoms in a short space of time.

Unless those who treat these patients recognize the importance of primary toxemias as bound up in symptom production in the case, these patients will continue to pass along from one to another. There is no more perfect result in ptosis work accomplished than, after the reconstruction work, or in association with it, attention is paid to the presence of an intestinal condition. It means permanency of the job or a result which is sustained for long periods of time.

GASTRO-INTESTINAL ATROPHY.

See Atonic Colon.

HEMORRHOIDS.

Hemorrhoids, like varicose veins anywhere, are most commonly caused by circulatory static states. In hemorrhoids, however, there is little doubt in my mind that infection plays a prominent rôle in their symptom production. The infection is usually of the aërobic type of organisms, and while no case of hemorrhoids has ever been cured by handling of intestinal toxemia, the presence of troublesome hemorrhoids is suspicion enough for biologic study.

HYPERACIDITY, HYPERCHLORHYDRIA.

The commonest origin of chronic gastric hyperacidity and hypersecretion is intestinal. The sooner the etiology of this commonest of all gastro-enterological conditions is recognized by specialists in this line, the sooner proper handling of these cases will be accomplished. As a rule in these cases proper attention in general ways is required, because, even though the intestinal toxemia be the exciting cause, once the stomach has been irritated so as to produce these states, even with the removal or marked benefit of the bowel condition, more or less continued functional mischief in the stomach will continue for lengths of time—sometimes several months.

ILEOCECAL INCOMPETENCY.

This has already been presented in connection with the section on atonic colon. There remains in addition to say that this condition represents a relaxation of the structures at the ileocecal valve, secondary to an intestinal toxemia. It requires only and always medical handling and never any surgery, no matter how many articles are written on the surgery of it, who the advancer is, or what type of operation is suggested.

INDICANURIA.

This, together with biluremia and other like conditions, merely represents one of the symptoms of an intestinal toxemia. Of course I except here instances of excess indican in the urine due to the resorption of body fluids, etc. Indicanuria is merely a symptom, in no sense an entity in itself.

INTESTINAL STASIS.

Much has been written on this subject from the surgical standpoint, and to submit cases of ileocolonic stasis to surgery means one of two things: first, that the medical handling over years of time has been most deficient and that surgery is necessary to accomplish benefit, or it means outright surgical exploitation. Intestinal stasis is a medical problem.

I except here instances where a stasis is essentially due to an obstructive or anchored condition. When, however, these do not exist or they do not exist in real dynamic ways, medical handling is all that is called for. The medical handling in the past or at least that which I see in this condition has been, on the part of practically all men, that of a simple type of diagnosing, in which no work other than an x-ray examination is engaged in and the diagnosis of stasis is made, the treatments being the use of diets, purgatives, etc., or, if the attendant be surgically inclined, by operation. Practically every case of entero-colonic stasis is definitely one of a chronic intestinal toxemia, and should be studied for a biologic diagnosis and treated accordingly.

NEUROTIC ABDOMEN.

The instances of neurotic abdomen are not uncommon in routine clinical work. They usually represent a complaining patient in whom after exhaustive examination no definite reasons for the complaints can be found. The individual, too, in presenting the complaints is usually hurried, anxious and inconsistent in manner and description of them, and the maze, variety and broad distribution suggest (when all examination findings are negative) that this diagnosis may be warranted. There are many primarily unstable types of people, and those who have become so. These in my experience are found more commonly among those who are born in Europe than here, and among certain races neurosis does seem to play a prominent part. However, it is not uncommon for me to see individuals in whom this diagnosis has been made and in whom I likewise believe it was justified, who on careful examination of the intestinal canal proved the presence of an intestinal toxemia, which, when removed, caused the disappearance of the symptoms of which the individual complained.

This includes all the various types of distress, painful areas here and there, sometimes quite persistent, and practically all that goes with such a diagnosis. I therefore feel that as a cardinal rule the diagnosis of neurosis should not be made until last of all, and, before it is made, intestinal studies should have

been engaged in as an explanation for its presence. As is true in all neuroses of course, the many well-known factors which are important in their production require estimation and attention. The reader is referred to the section on Neuroses.

OMENTITIS.

I do not wish to add to the nomenclature of medicine by dilating much upon the above term. There are conditions which, while vague and impossible to diagnose clinically, are those of congestive and inflammatory states of the omentum commonly with the production of adhesions and bands. What has been said in connection with peritoneal adhesions holds true in these cases, of which I have had three notable instances with ascites, where on the handling of an intestinal toxemia striking results were accomplished.

PROCTITIS.

In innumerable books and articles, the subject of proctitis, which is usually chronic, has been presented. But no one seems to realize that chronic proctitis, whether of the hypertrophic or atrophic forms, is secondary to a chronic intestinal toxemia, and that the handling of the proctitis is not a matter of fussing around with local treatments, but one of the study of the biology of the intestinal canal and proper biologic handling. Proctitis is a common condition in the human being. Anyone making routine examinations with a proctoscope will agree to this. In itself it is not a symptom producer, except that occasionally there may be discharges of mucus and perhaps moisture which collects around the anus and which may cause itching, etc. It represents one of the easiest types of examination to suggest the presence of an intestinal toxemia, and, as a rule, when the toxemia is properly and successfully handled, the proctitis disappears.

PRURITUS (ANAL).

There are a number of causes of anal pruritus, and its extension forms to the scrotum and vulva. In the vast majority of them I believe that they represent an infection of the

anal-muco-cutaneous tissues, this being mostly streptococcic in type, but sometimes due to parasitic forms of the *escherichia* and *staphylococci*. The condition suggests the presence of a chronic intestinal toxemia, and studies should be made in that connection. Speaking of the graver and more persistent forms, results from handling the cases biologically have been eminently satisfactory.

PYLORIC OBSTRUCTION.

There are instances of pyloric obstruction as well as obstruction in the lower part of the ileum, where the process is non-ulcerative and non-malignant, and represents a thickening of the tissues, usually with more or less hypertrophy of the muscular element with white fibrous tissue deposit. I have yet to see a case of these where an intestinal toxemia is not present, and I believe firmly that the conditions mentioned are resulting states from it. There have been seventeen cases of definite pyloric obstruction handled along the intestinal lines with distinct benefit to the local state of the stomach and ileum. The rule is, however, that when the obstruction is definitely established nothing medical to overcome it is of much or any avail.

PYLOROSPASM.

The vast majority of instances of pylorospasm are due to definite disease in the proximal portions of the stomach or duodenum, the gall-bladder, the appendix, or the pleural bases. There are, however, not a few instances of pylorospasm, as well as gastropasm and spastic states of the small intestine, that are bound up in resulting conditions or accompanying conditions with an intestinal toxemia. Care, of course, must be taken in diagnosing, but when no definite pathological cause is believed to be present, biologic studies of the intestinal canal and proper handling are in order.

REDUNDANT COLON.

Redundant colons are largely congenital with resulting states of constipation, usually with an atony and a dry colitis,

and these should be handled along these lines rather than always by surgery. Intestinal toxemia should always be sought for, and when present, proper treatments engaged in. As in dilatations of the hollow digestive structures, redundancy or attenuations are only partially restored to normal when the toxemia is controlled. But in this connection it must be remembered (excepting in dilatations proximal to obstructions) that by proper handling, relief of local distress and symptoms and improvement in general health are usually accomplished.

SPASTIC ANAL SPHINCTER.

What is mentioned in connection with spastic colon is somewhat in order here. However, there are instances of spastic anal sphincter caused by local pathology in the bowel, occasionally this being one of the forms of proctitis which requires attention. Ofttimes, though, individuals are seen with a chronically tight sphincter in whom the condition represents the results of a spastic colon, and since spasticity of the colon is largely bound up in intestinal toxemia, the solution for the anal difficulty would be found in attention directed to the infection in the bowel. There are several causes of this condition and all should be evaluated in the case.

SPASTIC COLON (CONSTIPATION).

Everybody seems to be concerned about spastic colon being a neurological condition. Nobody seems to be getting anywhere with regard to it, and largely the cases go on continuously. Spasticity of the colon and more or less irregular haustral contractions are a common resulting state from intestinal toxemia. Usually the bacteriology represents fermentation groups and is definitely local in its irritation and results. Commonly these people have more or less disturbance in the vegetative nervous system, but largely this disturbance is secondary to the intestinal disorder. When the profession learns to handle spastic colitis or spastic constipation along biologic lines, the subject will cease to be as debatable as it is at present.

SPASTIC RECTO-SIGMOID APPARATUS.

What has been said in connection with spastic colon is true in this. It merely represents a localized part of the colon.

ULCER (GASTRIC AND DUODENAL).

The commonest causes of these ulcers are the lodging of bacteria in the submucous venous plexus carried there by the blood, the organism originally coming from the intestinal canal. There then occurs a localized infective phlebitis, a damming of the circulation, and, the tissue being dead, is digested away by the gastric juice. When an ulcer is once produced it makes but little difference where the organism originally came from or what it was. The local infection is over and no amount of intestinal handling is going to make any difference locally. But, in the absence of other reasons, a case of ulceration may require intestinal study either because of recurrence of symptoms or the x-rays have shown that the ulcer had been healed and that it had reopened or new ulcers had been formed. From these standpoints, the biology in the intestinal canal may be important in the case and the case may require handling in this connection as well as what may be necessary locally to encourage the ulcer to heal. As a rule, in chronic ulcer, especially when of some years' standing, biologic treatments are of no curative value.

CHAPTER XVIII.

CARDIOLOGY.

Angina Pectoris—Coronary Sclerosis—Endocarditis—Functional Heart Conditions—Hypertensive Heart Disease—Myocarditis—Nervous Heart—The Cardiac Irregularities—T-Wave Negativity.

ANGINA PECTORIS.

See Section on Internal Medicine.

CORONARY SCLEROSIS.

See Angina Pectoris in Section on Internal Medicine.

ENDOCARDITIS.

See Section on Internal Medicine.

FUNCTIONAL HEART CONDITIONS.

Leaving the arrhythmias and the nervous heart cases out of consideration, there is left a group of conditions, such as cardiac consciousness, cardiac pain, pseudo-angina, irritable heart, effort syndrome, palpitation, and functional heart murmurs. The latter are usually of the mural type, sometimes due to the anemia, but more especially to slight grades of cardiac insufficiency of muscle tone or relaxation of the valve rings. As a group they are seen constantly in chronic intestinal toxemia and generally disappear on improvement with proper handling.

HYPERTENSIVE HEART DISEASE.

This is an entity term in common usage which should be discontinued. It represents duplicate or modified nomenclature and is misleading. It may here be mentioned that the arteries, heart and renal structures are all bound up as cousins in disease, the same factors bringing on disease in all of them at the same time, although often different in degree in the different organs, and, therefore, the symptoms and symptom

groups vary somewhat in individual cases. Consequently, such terms as the above, and "cardiorenal disease" and "cardiorenal vascular disease," while they may fit appropriately in certain instances, are really confusing in relation to treatment. This whole group is so intimately bound up in intestinal toxemia of chronic type as their cause, that the men interested in so-called cardiology must recognize this for the best understanding of its pathology and therapy.

MYOCARDITIS.

See Section on Internal Medicine.

NERVOUS HEART.

The designation above is used because it is the most frequent of all heart conditions, is easy to handle properly, and often is neglected until serious cardiac disorder eventually becomes established. The disturbances of the afferent and efferent nerve branches in their effects upon heart action, pain production or other abnormal sensations, of course can and do often present themselves as a pure neurosis or from bad routine of life. In the vast majority of instances, however, the neurosis is assumed, when in fact this neurological condition is only a resulting state from an intestinal toxemia, which if not existent in the individual there would be no neurosis. It is my opinion that after making the physical, x-ray, and electrocardiographic examinations—when these are normal—biologic examinations are called for before the case is dispatched as of no special interest or requiring no special attention.

THE CARDIAC IRREGULARITIES.

The heart walls contain a large number of nests of specialized neuromuscular tissue. Bound up with these are the sino-auricular node, the auriculo-ventricular node, and the bundle of His. The subendocardial network of nucleated specialized cells are in close relation with the heart muscle fibers, and the whole system receives its supply of energy from branches of the vagi (inhibitors) and the sympathetics (ac-

celerators). These two sets of physiological antagonists preside over heart rhythm, in which the neurological structures of the heart proper require "clear lines" for normal function. There is no more common finding clinically than disturbances of rhythm in intestinal toxemia cases.

Sinus Arrhythmias.—This is a common finding in which the toxic effects are on the sino-auricular node or on the ganglia and nerves of the cardiac plexus back of it. There are other toxic factors that may produce it, such as those of cholemia, uremia, pregnancy, etc., but those due to intestinal toxicity comprise the vast majority of clinical cases seen. Leaving out of consideration the emotional, respiratory and phasic forms, that most commonly seen is of the sino-auricular block type in which bradycardial attacks play off and on perhaps. As a rule these are controlled by the intestinal treatments.

Auriculoventricular Heart Block.—Mild forms of the condition suggesting delay at the auriculoventricular junction, playing off and on in graphic record studies are not uncommon, and usually recover on treatment. Where, however, a permanent block of 2:1 ratio exists permanent mischief has taken place. The future of this subject is in the diagnosing of the earlier, functional, or transitory forms, and the possible control or prevention of the severe grade. When found—if acute infections of extreme grades, digitalis administration, and the rare congenital form can be excluded—biologic intestinal studies should be engaged in with appropriate treatment. The latter statement also includes the instances of dropped ventricular beats.

Extrasystolic Arrhythmias (Premature Beats).—These are common findings in cardiac irregularities and unfortunately too often are allowed to go on to degeneration of the auricle walls and thus become permanent. Whether the premature beats are believed to be from the auricles or the ventricles, in the absence of definite advanced heart disease, intestinal toxemia should be examined for.

Tachycardia.—This is the commonest of all the arrhythmias seen in intestinal toxemia. Of course the nervous

and emotional types must be excluded in the diagnosis. In the true paroxysmal types the same cause operates in the production, to which, of course, must be added the pyrexia cases and so forth. The hyperthyroid cases are generally intestinal toxemic in origin. Where distinct degeneration has occurred in the auricles, the condition is beyond permanent control.

Auricular flutter and auricular fibrillation represent, as a rule, an advanced type of cardiac disease and these are almost wholly only digitalis cases. When definitely established, no benefits on the heart condition can be expected from intestinal toxemia therapy.

Combined arrhythmias are of different varieties, such as auricular flutter with extrasystoles, heart block with ventricular extrasystoles, etc. In them the judgment of intestinal toxemia significance depends upon the clinical findings and beliefs in each case.

T-WAVE NEGATIVITY.

In all modifications of the Q R S complexes in which the electrocardiographic changes parallel changes in the size of the right and left ventricles, biologic studies are called for to explain one of the causes. Especially is this true in types of T-wave inversions. In these, of course, are the definite hypertension, the probable preëxistent hypertension, and the hypertension with coronary sclerosis, as well as the instances in which it occurs in aortic and mitral valve lesions and the more straight myocarditic types.

As a rule when the cardiac changes are well established but little can be accomplished in the way of benefit on the heart condition. Here and there, however, in a case in which the findings of significance are more electrocardiographic than those found on physical examination or complaint of patient, intestinal studies should be engaged in, because, not infrequently, much benefit can be accomplished, or at least its proper handling would save the heart from rapid extension of disease. I have seen no cases in which blanking of the T-wave has recovered a normal tracing, but considering the high mortality rate in these persons, not a few that had been handled biologi-

cally for an intestinal condition have lived on with comfort for years.

There are so many cardiac lesions found at autopsy in which the intestinal state can have to do with their production, that one needs but to record them in part here. They represent dilatation of valve rings; sclerosis of vessels and valve flaps; thickening of valves; puckering of valves; binding together of cusps; vegetations and fibrous thickening of flaps; calcification of flaps; scarring of flaps; stenosis of flaps; fusion of aortic commissures; fish mouth stenosis, etc. A suspicion of cardiac disorder—syphilis excepted and rheumatism included—means that these biologic studies should be engaged in. In late and well-established cardiac disease, as a rule, no time or expense need be wasted, the treatment being digitalization and symptomatic.

At the present time there is a lively interest in cardiac diseases and conditions, the mortality rate of which disorders seems to be rising rapidly. Clinics are being established, organizations formed and specialized types of medical periodicals advanced. The work engaged in today is mostly that of diagnosis of cardiac lesions and but little of etiology and prophylaxis. The treatment of cardiac conditions is rather limited in scope and not highly curative in percentage of permanent results. If prevention is properly the standard of this era of medicine, and I believe in it fully, then I fail to see how the biologic side of medicine, and especially of the intestinal canal, can be neglected in connection with cardiac disease.

CHAPTER XIX.

NEUROLOGY AND PSYCHIATRY.

Anxiety States—Depressions—Epilepsy—Functional Mental Symptoms—Hysteria—Manic-depressive Psychoses—Menopausal Neuroses—Migraine (Hemicrania)—Neuralgia—Neurasthenia—Neuritis—Organic Neurology—Psychasthenia.

CAUSES OF NERVOUS AND MENTAL DISEASES AND DISORDERS. SOME BELIEFS ON PSYCHIATRY, THE NEUROSES, AND EMOTIONAL STATES.

If the reader will look back on page 138 in the schema on constitutional and environmental make-ups he can get some worth-while idea of the factors which pertain to the production of neuroses. Practical experience and observation have taught me, when the individual with functional nervous and functional mental disorder is considered more along the lines of an individual who is bodily ill, chemically, toxically, organically, or in some definite way, and one in whom benefits from treatment could come from the handling of these body conditions, that largely the academic superstructure of psychiatry falls into insignificance excepting where there are fixed conditions, like hereditary factors, advanced stage, race, etc.

Practically all psychiatrists today depend upon two exciting causes for explanations and handling of mental disorders. In practice and literature they are expressed as: *Physical*—toxic, of which the most prominent of the exogenous are alcohol and syphilis, the drug addictions and the metal poisons, together with those ensuing from some of the infectious diseases; and of the endogenous there are mentioned just a few words or a few lines which suggest that they might arise from the gastro-intestinal tract, or be the result of a chronic nephritis, myxedema, exophthalmic goiter, etc. Other physical causes are the traumatic, exhaustion states, and body disease.

The second important exciting causes are agreed upon to be *mental* in origin, such as worry, shock, fright, horror, anxiety, grief, suspense, fear, etc.; these largely weaving in the domestic, social, religious, or financial status of the person. These we may all accept as facts, but the most important cause of all is the endogenous poisons which originate from the gastro-intestinal tract, and on this point neurologists gloss over the subject with simple and pointless statements and go no further, even in the way of suggestion of therapy for it. The fact is that in these five thousand instances of chronic intestinal toxemia, over eleven hundred had had experience with neurologists, psychiatrists, and in sanitariums, etc.; instances of patients where the conditions were functional and where in practically all a perfect result was accomplished so far as the mental state was concerned. Now if one were to judge such people, as is generally done, as being sick above and not below, why is it that by handling the intestinal condition the mental state cleared up? This, of course, excepts those cases that have fundamental causes such as heredity, etc., but does not except all those of so-called "acquired predisposition." My experience leads me to believe that a good biologic type of gastro-enterologist is an exceptionally good psychiatrist and neurologist when it comes to matters of therapy, and one who would accomplish far better results than those being brought about by the attitude as to causes, and methods of diagnosis and therapy in vogue today.

Psychiatry is designated as "that branch of medicine which deals with mental disorders, their causes, prevention, and treatment." It is one of the recent branches of medicine, an offshoot from neurology proper, and represents an effort to make concrete this part of neurology in some sort of fundamental way. To a clinician, like myself (who has dealt with these somatic disorders of the human being for thirty years, having seen many with various mental phenomena, all more or less understood from practical standpoints), to attend the clinics, listen to discussions by psychiatrists and read the various books on the subject, brings me to the conclusion that today there is a great deal in psychiatry that is ephemeral,

academic and non-practical. Especially is this true in the understanding of etiology connected with mental phenomena.

One can quite readily agree that heredity represents an important factor, and this with the Mendelian theory is rather a concrete portion of this subject. One can also agree that among the essential causes are drug addiction, syphilis, head injuries, acute and chronic infectious diseases; as well as the contributing or incidental causes of alcoholism, pregnancy, abortions, childbirth and lactation, worry, grief, anxiety, suspense, etc. Also there are factors in different races of people, as well as ages of individuals, environments and occupations in which individuals happen to be set. But on the other hand, trying to judge the symptoms of the disorders of perception in their many types and kinds, ideas, disorders of judgment, disorders of affectivity, etc., commonly leads one into a maze of impractical matter, especially when the disorders now classified under the neuroses are being considered.

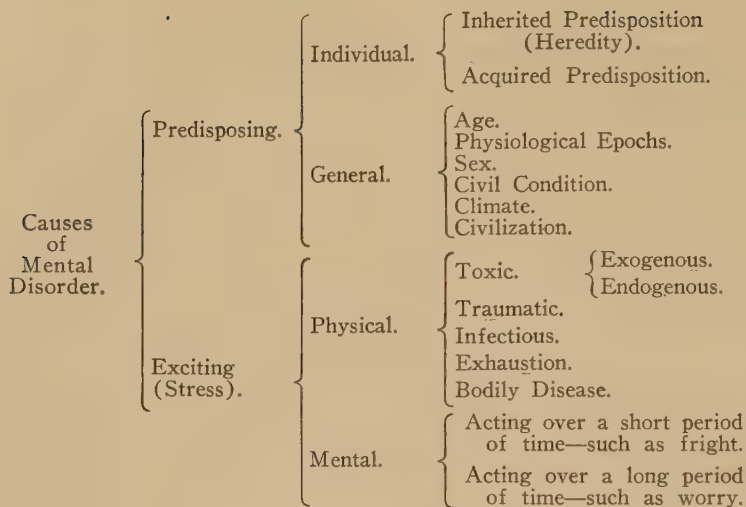
Finally, in the generally accepted practice, when psychology (an ever-shifting and most debatable subject in practically all of its detail) is accepted as a part of therapeutics, to which are added institutional commitment, the cold douche, the warm bath, isolation and symptomatic medication as the only methods of treatment—or these plus suggestion and psycho-analysis (in which almost everything is somehow sexual according to Freud), and dreams are interpreted and methods of procedure engaged in which on the face of them and by their very nature suggest the inability of handling these disorders—one must agree that the work is most academic, and far from the types of real fundamental medicine upon which practically all of the other specialties are based.

Among the psychiatrists and neurologists with whom I am acquainted are found various types of men: first, a neurologist who practically pays no attention to psychiatry, cares little about it, and is quite critical about the whole field of work; second, the neurologist or psychiatrist who is more of an endocrinologist than either one, and who if he did not depend upon common sense and other therapeutic measures would practically accomplish no results worth while; third, the neurologist or

psychiatrist whose principle of work is practically to make thin people stout, and stout people thinner, listen to their troubles and undertake to show them the folly of continuing with them, to give purgations in constipation, astringents in diarrhea, and *in toto* practically follows a common sense sort of therapeutics as all that is essential in the therapeutic handling of the entire subject; and fourthly, the ephemeral, academic type, who believes totally and practices fully psycho-analysis, believing that this is the main part of the entire treatment.

To my mind, as a simple physician, psychiatry has extended in theory and assumption so far from practical medicine that it views the person almost entirely from the head down, rather than from the body up to the head. It may be in the years to come that what is believed as the bases of psychiatry today will develop into a full, normal, practical and useful branch of medicine. But in my humble opinion the handling of the individual along the lines laid down today starts nowhere, is by no means practical, and usually gets nowhere. I recognize that these statements of mine will be met with severe censure, and that illustrative cases will be presented to show the error of my belief. I am willing to accept the fact that results have been accomplished here and there, but that they have been accomplished by therapeutic measures of psycho-analysis, rather than being totally accomplished by auto-suggestion of the individual and the suggestions of the medical attendant, is most questionable to me. I doubt very much that a psycho-analysis conducted with the patient some distance from the analyzer, the only means of communication being a telephone, could result in any worth-while benefit.

For these reasons I am going back and will adhere to my original teachings in medicine in the use of entity terms, etc., since these represent to me a more practical handling of these conditions by the busy clinician. According to White the causes of mental disorder are presented in the following classification, and this to a physician, a neurologist, and even a psychiatrist, is an excellent schema by which to be guided:



ANXIETY STATES.

The so-called anxiety neurosis of Freud, anxiety hysteria, and some of the functional other types are commonly seen as a direct result or one in which intestinal toxemia acts as a contributory cause.

DEPRESSIONS.

If those present in the pre-senile psychosis and the psychogenic type, or the distinct psychopathic types of individuals can be excluded, the rest are often associated with intestinal toxemia of various forms.

EPILEPSY.

Of these there have been 37 cases, most of them of the grand mal type. There were at least twice as many more which I believed unsuitable for treatment. Since epilepsy at times will cease or modify without any treatment, it is not just to claim benefit from any type of treatment. Of those that have ceased having attacks there were several, but what is most significant is that throughout all of the cases handled the attacks became fewer and fewer, and farther and farther apart, and, while the majority have an attack occasionally at long in-

tervals of time, the results in the handling of this subject from an intestinal standpoint were most remarkable. The diets suggested in the previous chapters were used and in no instances any special types of diet, such as high fats, etc. The only other measures employed besides dietetic and biologic treatments according to the intestinal toxemia present, were transduodenal lavages with hypertonic saline solutions and small doses of luminal. *Petit mal* is more difficult to control.

FUNCTIONAL MENTAL SYMPTOMS.

The above term is used for the want of a better one to describe a number of mental phenomena different individuals may have and which cannot be associated with disorders of psychiatric or neurological nature, but which nevertheless play more or less continuously on otherwise normal persons, sometimes much to the distress of the individual and oftentimes leading to error in neurological and psychiatric diagnoses. They comprise a vast array of mental symptoms such as disorders of the will, slight grades of hallucinosis, degrees of amnesia, errors of attention, complex defects, disordered judgment, depressions, disorientation, distractability, dreams, errors of reference, illusions, incoherence, periods of melancholia, abnormal reaction, suggestibility, vertigo, etc.

In all of the above before the individual is dispatched as peculiar or eccentric, and even in the instance of definite neurological diagnosis, it is advised that a biologic examination and diagnosis of an intestinal status be made and, when proven, therapy therefor instituted. The human brain in its functions called mental is most easily disturbed by toxic factors, especially if these be chronically present. Of these factors the most important and most common is found in biologic errors of the intestinal canal.

HYSTERIA.

When this can be differentiated from epilepsy I am inclined to believe that intestinal toxemia can be one of the exciting causes. However, the average hysterical individual is dispositionally that way and one must be careful in promises by

biologic treatment. If care is exercised in the selection and examination of the cases, some striking results will be met with.

MANIC-DEPRESSIVE PSYCHOSES.

The main etiology of this condition, in my opinion, is that of heredity in which the endogenous causes, such as alcohol, are commonly found, and in which in addition intestinal toxemia is undoubtedly a contributing factor operating in some cases at the time of acuteness of symptoms. I have seen nine instances of this type, none of them actually cured, but the shortest had a two-year interval between attacks—being perfectly well during that time—and the longest nine years. In this connection it must be mentioned that in all of the manic-depressive psychoses great care should be exercised in the general examination for visceral disturbances. In the majority of cases, however, these are not found, but in careful biologic work, foci of infection, usually in the intestinal canal, and most commonly those bound up with some of the forms of streptococci, will be found as the most important etiological factors.

MENOPAUSAL NEUROSES.

While the majority of these are more particularly bound up with the menopausal phenomena, there are not a few in which an intestinal state can be a distinct contributory cause.

MIGRAINE (HEMICRANIA).

In all of the instances of chronic intestinal toxemia where distinct hemicrania was present no marked benefit was accomplished, only a partial one here and there. I, therefore, am inclined to believe that hemicrania is a hereditary condition not capable of much, if any, sustained benefit by intestinal treatments.

NEURALGIA.

While exposure, local infection, or injury are responsible for many instances of neuralgia, there is no doubt that recurrent neuralgia may be due to intestinal causes. Of these I have

seen instances of the sphenopalatine ganglion, glossopharyngeal, occipital, brachial, intercostal, lumboplexus, and the sciatic forms. In instances of recurrence of these conditions, and they are not uncommon, studies of the intestinal canal are called for.

NEURASTHENIA.

This rather common neurological condition is now classified under the psychoneuroses, but the author prefers to term it "fatigue toxic neurosis." It has been my observation that once a neurasthenia is well established, even with the correction of an intestinal condition, months of time are required for the disappearance of the symptoms, and during this time tonics, rest, baths, freedom from work, etc., are called for. But the neurasthenia that is due, either in a primary or in a contributory way, to a toxic intestinal state is not cured as quickly by the means generally used as when at the same time with these the intestinal toxemia is controlled. And more than that, the rule in cases so handled is that there is no "red herring" of neurasthenia or semi-neurasthenic states continuing on the trail for years afterward as is so commonly seen following the measures of treatment employed today.

Of these cases there have been over four hundred, practically all in the individuals who have gotten entirely well without sanitarium treatments or withdrawal from home, business, etc. It is my belief that the commonest causes of fatigue toxic neurosis are states of chronic intestinal toxemia.

NEURITIS.

A peripheral nerve may become diseased from toxic products from the intestinal canal as well as from other toxic causes. This includes cases of non-alcoholic multiple neuritis or those from other definite toxic reasons. Of such instances there have been several intestinal in origin.

ORGANIC NEUROLOGY.

In the author's cases the instances have been few of definite forms of organic neurology in which studies of the intestinal canal were made. In practically all of these the neurological

diagnosis was definite and although an intestinal toxemia was present, it was not possible to separate the cause from effect, and it was usually judged that in the presence of the definite neurological condition no benefit could ensue by biologic treatments of the intestinal canal. However, there have been enough instances to suggest a strong possibility of intestinal toxemias being etiological factors in the production of organic nerve disease. And the author here merely wishes to suggest that in instances of definite organic disease biologic studies be made with the hope of finding out to what extent and in just which disorders intestinal toxemias are important etiological factors.

Among these that I would suggest to be studied particularly are: multiple sclerosis, combined degeneration of the cord, Friedreich's ataxia, amyotrophic lateral sclerosis, the myotrophies, progressive spinal-muscular atrophies and dystrophies, syringomyelia, Landry's paralysis, myelitis, various plexus nerve inflammatory lesions, myasthenia gravis, the bacteriology of brain abscesses, the cerebral cases in children, paralysis agitans, Wilson's and Westphal's diseases, chorea, as well as some of the disorders of the cerebral nerves.

PSYCHASTHENIA.

What has been stated above in connection with neurasthenia proves to be even more true in this type of so-called psychoneurosis. I refer in my cases to those who have not had fixed emotional obsessions, such as phobias, morbid desires, etc., and in whom there is a distinct lowering of the psychological tension, a status of affairs mental as compared to neurasthenia in a bodily way. To me psychasthenia is most commonly seen as a neurasthenia affecting the emotional centers of the brain, rather than dysfunction of nerves distributed through the general body, as seen in neurasthenia. The emotional spheres of the brain are most sensitive to endogenous toxins, and in the production of irregularities in them chronic intestinal toxemias are often not only primary factors, but generally they are the most important of the contributory ones. See Neurasthenia.

CHAPTER XX.

OTORHINOLARYNGOLOGY.

Deafness—Hay-fever—Labyrinthine Disturbances. Vertigo (Intestinal)—Laryngitis, Chronic—Nasopharyngeal Conditions—Purulent Chronic Middle Ear Disease—Rhinitis, Acute Colds in the Head, Chronic Rhinitis—Sinus Disease, Chronic and Recurrent—Tonsillitis, Acute, Chronic, and Purulent—Tracheitis, Chronic.

DEAFNESS.

Tinnitus is a frequent symptom in neurasthenia from bowel states. In what may be termed the neuritic, intermittent, partial and catarrhal types of deafness, the local condition in the nasal mucosa may show an extension of the process up the eustachian tubes (chronic eustachitis) and when due to bowel states; direct or contributory, an intestinal state may require attention before definite local and sustained benefit can be brought about. While in most instances otosclerosis is hereditary or at least occurs so early in life that it appears to be, there is a possibility that the spongy condition of the bone of the labyrinthine capsule may be bound up with intestinal toxemia starting early in life. When the disease is well established no benefit from bowel treatments has been brought about.

HAY-FEVER.

See Section on Internal Medicine.

LABYRINTHINE DISTURBANCES. VERTIGO (INTESTINAL).

Intestinal toxemia, especially when associated with distinct intestinal stasis, seems to be accepted by many of the specialists in this connection, although there are not a few who do not seem to recognize it. Vertigo has already been mentioned, and when Ménière's disease can be excluded, intestinal toxemia should be examined for. On the slightest suggestion of ver-

tigo, nystagmus, or disturbance of equilibrium, in the absence of evident causes, intestinal studies should be made.

LARYNGITIS, CHRONIC.

Chronic hyperplastic, simple catarrhal (diffuse), subglottic, chronic perichondritis and chondritis in many instances require studies of the intestinal state. When local causes are controlled the persistency of these conditions may be due to intestinal toxemia in which the bowel state is an important contributory factor. Chronic atrophic laryngitis occupies the same significance as that mentioned in connection with ozena.

NASOPHARYNGEAL CONDITIONS.

In the simple chronic, lymphatic engorgement and enlargements, in some of the polypi cases and persistent congestive states, the bowel may be an important source of the continuation of these conditions.

PURULENT CHRONIC MIDDLE EAR DISEASE.

No special significance is attached to the bowel in the acute cases. In the chronic forms in the young as well as in adults, since this disease often shows signs of chronicity almost from the beginning, and an important factor for its continuation can be a bowel state, biological studies are in order in many of the instances.

RHINITIS, ACUTE COLDS IN THE HEAD, CHRONIC RHINITIS.

Persons who are susceptible to repeated seizures of acute coryza may be susceptible to certain infections, there being little or no local or general resistance to them. When these infections are those of the local head organisms (*Micrococcus catarrhalis*, *Bacillus fusiformis*, influenza bacillus, etc.) the bowel may or may not figure as an important contributory factor. There have been many instances in which benefit of an intestinal toxemia has been a better preventive of "colds in the head" than the use of immunizing vaccines or antigens therefor.

While there is some doubt in instances of the importance of intestinal toxemia in the acute conditions, there is very little in the chronic. In simple chronic, hypertrophic, and the purulent forms, these chronic bowel conditions are commonly direct causes of trouble. There are many instances in which chronic states exist continuously, sometimes more, sometimes less. The enhancement of the toxic bowel state which is found during the winter in our temperate climates and especially in those dwelling in large cities proves that the head condition follows that in the bowel. Many persons having these head conditions need but a few weeks of being shut up in apartments and houses in cold weather, thus getting less physical exercise, sunshine and outdoor air, as well as eating less of the summer types of foods, and increasing the protein intake, to have the toxic state in the bowel more intensified, and the condition in the nasal fossæ promptly responding.

There is no more conclusive fact in all of this work than to see the remarkable benefits that are brought about by intestinal treatments in many of these head conditions. No case, however, of true atrophic rhinitis (*ozena*) has ever been benefited distinctly. It is probable here that the mucosa is too far gone for restitution. Especially interesting in the way of benefit have been the cases of "chronic sneezing" or rhinitis hyperæsthetica. Some of these are due to straight food anaphylaxis, but in the majority, the anaphylaxis is secondary to the bowel state and the sensitive mucosa may be due to the toxemia without anaphylaxis. In vasomotor rhinitic disturbances studies should be made.

SINUS DISEASE, CHRONIC AND RECURRENT.

Sinus disease is divided into the acute and chronic forms, and, according to location, the maxillary, frontal, ethmoidal (anterior and posterior) and sphenoidal. More than one sinus may be diseased at the same time. The acute processes are generally those of short duration, due to infections of the upper respiratory tract by bacterial organisms capable of producing such. Among these are commonly the *Micrococcus catarrhalis*, influenza bacillus, Vincent's spirillum, smegma ba-

cillus, etc. Such conditions are usually self-limiting, although they may be followed by ulceration, bone changes, or by chronic processes. Acute as well as chronic infections are due most commonly, however, to organisms which are denizens of the intestinal canal. Of these are the various streptococci, pneumococci, staphylococci, and the *Escherichia*.

All the specialists in nose, throat and ear disorders believe and teach, and their text-books claim, that infections of the sinus, nose, ear, throat, and larynx are due to local inoculations. This is no doubt true to a great extent, but it does not take into consideration that such infections and organisms are commonly blood-borne to these structures, that the intestine in the case generally shows the presence of the same organisms, which, though the head infection is improved or cleared, generally persist in the intestine.

Throughout all of sinus disease the incidence of "latent infection" is a most important factor. In outspoken infection the interest may be justifiably local (not always), but since throughout all of sinus infections persistency and recurrence are important factors, a general significance of infection attaches to them. That bacteria are easily carried to the head sinus can be accepted as true. These do not originate always in the gall-bladder, appendix, pelvis, kidneys, etc., but come more frequently from the intestines. In an instance of chronic, persistent, or recurring sinus disease, when organisms, either in straight or associated infections, are intestinal in classification, the bowel should be thought of as a source requiring attention together with whatever may be done locally in the head sinuses.

What must be remembered here, too, is that the head condition may be of the non-suppurative as well as the suppurative type in which this figures. An original infection may become sterile from the disappearance or there being too few of the original organisms left to obtain positive cultures from local swabs. These negative results do not exclude the significance of the bowel as an important factor. The original infection may still be present but much attenuated, reinfection may take place, and the toxins from symbiotic groups to the original

infection in the bowel can, and often do, keep the head process going or interfere seriously with recovery. In all the chronic cases, whether the sinus ostium is closed or open; in the hyperplastic, whether with retention or only thickening or both; in polypoid conditions, whatever the local bacteriology is or is not, intestinal studies will often do much to clear away many of the doubts, and attention to the intestinal condition will be the solution for best results in therapy.

TONSILLITIS, ACUTE, CHRONIC, AND PURULENT.

In acute forms of tonsillitis it is most probable that the infection gains entrance into the tonsillar tissue from the surface. In the chronic hyperplastic, often in the lacunar, and commonly in the various infectious types, the intestinal canal should be thought of as a source of infection. Added to this, when systemic benefits are not forthcoming or are too trivial after tonsillectomy, the gut condition should be studied.

More chronically infected tonsils occur from the intestines than from the surface, and more chronic infections persist in tonsils because of infectious bowel conditions than are suspected. A last truism is that infection in tonsils (and other head sources) does not cause infected bowel states to the extent that is believed, neither are the latter cured by the removal of the head infection.

Cultures removed from interior of tonsils show an incidence of hemolytic streptococci as high as 90 per cent. with a few Pfeiffer, staphylococci, pneumococci and fungi. These positive cultures are often obtained in tonsils that show no gross evidence of pathology, no softening, no pus, and only a little and inconstant increase in connective tissue and inflammatory cell deposit. Commonly only a few wandering cells are seen. In a broad way it is logical to assume that these contained organisms were originally surface in lodgment. But considering the high incidence in which organisms are found in the mouth and throat, the common absence of tissue pathology (especially the inflammatory or resulting inflammatory types seen in infection generally in the body), and the constant

washing of tonsil surfaces with swallowed foodstuffs and fluids, it is also logical to believe that these organisms get into the tonsils from the body side—the tonsillar tissue being good material for lodgment and then there occurs continuation of infection, latent or chronic.

TRACHEITIS, CHRONIC.

What has been stated regarding the larynx holds true here.

CHAPTER XXI.

OPHTHALMOLOGY.

Amblyopias—Blepharitis—Cataract—Choroiditis and Choroidoretinitis—Conjunctivitis—Episcleritis—Errors of Refraction—Iritis—Keratitis—Neuroretinitis—Ocular Manifestations of Nervous Diseases—Optic Neuritis—Postoperative Infections—Retinal Diseases (Retinitis)—Scotoma (Central, False, Toxic and Scintillating)—Uveitis—Vitreous Conditions.

AMBLYOPIAS.

These comprise a group of cases in which, with the usual toxic causes (alcohol, drugs, tobacco, etc.), the intestinal canal should be considered. There have been so many of these, even among my own assistants, who have recovered on intestinal toxemia treatments, that there is little doubt that those conditions of the bowel are most important factors.

BLEPHARITIS.

The exudative disorders of eczema, urticaria, rosacea, etc., suggest intestinal study. This includes especially herpes of the lids or of the cornea (ophthalmic herpes). Not a few instances of blepharitis marginalis were seen to benefit markedly on intestinal treatments, the same being true of several instances of intense blepharospasm.

CATARACT.

Not a few of the specialists believe that the intestinal canal is the commonest cause. On this, conclusion is difficult, but in the grey or simple type it is very probable. However, when a cataract is well established, intestinal treatments are of no avail to stop it, although there have been a dozen or more cases in which no further advance in the cataract was noted.

CHOROIDITIS AND CHOROIDORETINITIS.

When the congenital, tubercular, tumiform and syphilitic types and causes can be eliminated, choroiditis and choroidoretinitis should have intestinal studies. I have seen not a few

instances of exudative, disseminated and central choroiditis stopped and controlled by such handling.

CONJUNCTIVITIS.

In atrophy, brown spots, recurring acute and chronic hyperemia, and simple catarrhal and gouty conjunctivitis, the intestinal tract should always be thought of as a cause. Particularly is this true in the chronic dry type of which there have been not a few in my series.

EPISCLERITIS.

This condition, due to exudate into the episcleral tissue, when not due to nasal obstruction or the rare instances of syphilis or tuberculosis, is generally due to intestinal toxemia. The generally accepted "gout" or "rheumatism" types are in this group. The same is true of the fungous and the deep sclerotic inflammations.

ERRORS OF REFRACTION.

These conditions are so common that it would be easy to assume that errors in the intestinal canal contain factors of importance when both are found in the same individual. Therefore, the author hesitates to link cause and effect in these connections. The eye as an optical apparatus is so dependent on depths of focal lines, curvatures, symmetry, etc., that it is most possible in continued usage that errors of refraction could occur for no definite systemic etiological reasons. As a rule, too, when any of these errors is established, it usually is so in a permanent way. There have been so many instances of claim on the part of ophthalmologists of betterment leading to the use of "less strong glasses" that interest was constantly awakened, but definite cure practically never occurred. In this group, too, similar benefits have come about in non-toxic cases so that it is difficult to be definite. It is possible that here and there in refractive errors intestinal toxemia studies might be made; especially would this be true in early presbyopia and in asthenopia.

Having a refractive error and headaches, the majority of ophthalmologists are prone to the opinion that headaches, head pains, or functional eye conditions are due to refractive errors and that its correction with glasses would or should cause relief. Experience proves even when the refractive error is minor in degree, that this is in no sense uniform. There are symptoms of intestinal toxemia similar to those found in refractive errors, and there are instances of refractive errors in toxic individuals in whom these symptoms are due to the toxemia in part or wholly, irrespective of the eye condition. I need but to state that there have been many who have had glasses prescribed for continued use for the relief of distressing complaints, who, after intestinal handling, had no further need for them. The author wishes the profession to understand in this statement that correction of an intestinal condition is in no sense a curative treatment of errors of refraction.

IRITIS.

Inflammations of the iris, when of the serous, plastic, dysenteric, relapsing, or the vague forms of the acquired types, always require intestinal studies, and biologic errors will be found to be the common causes. The tissues of the iris are particularly sensitive to intestinal toxemias, continuation of which in these cases should be eliminated when possible.

KERATITIS.

Whether involved with the iris, the sclera, or only of the cornea and in all of its many forms, a keratitis is as often endogenous as it is exogenous in infection. For this reason, and because of its serious consequences to eyesight, intestinal studies should be made.

NEUORETINITIS.

There is no more hopeless etiology than that connected with the above serious disorder in other than the brain tumor cases. In the so-called papillitis or intraocular forms especially, the possibility of intestinal toxemia should always be thought of even when the symptoms seem to be associated with the clin-

ical conditions of sinus disease, focal infections in the throat, ear, etc., and renal disease. There is no doubt that control of the extension of the process has occurred often enough to bring the intestinal canal into prominence as a contributing cause, at least.

OCULAR MANIFESTATIONS OF NERVOUS DISEASES.

Since many nervous disorders are due to intestinal toxemia it follows that correction of the latter may be necessary for the control of eye conditions. Among these eye troubles are the involvements of motor nerves (third, sixth and seventh), unexplained miosis, mydriasis, hippus, hemianopsia, hemiachromatopsia, and slight grades of color blindness, especially the more functional types, exophthalmic goiter, organic and functional nerve disease and disorders, etc. The same is true in some instances, perhaps, of the sympathetic eye diseases, although usually no opportunity for research work is possible, at least, not in those I have had to do with.

OPTIC NEURITIS.

What has been said in neuroretinitis is true here. In all of the infectious forms and the retrobulbar type, intestinal toxemia studies would be in order.

POSTOPERATIVE INFECTIONS.

Not all infections following operations on the eye are due to local inoculation. The surgeon need not be conscience-stricken on this as to his technic, etc., because they often occur from the blood stream in intestinal toxemia cases.

RETINAL DISEASES (RETINITIS).

The most sensitive tissue of the eyeball to toxic states is the retina. Often these states are secondary to circulatory disturbances (such as seen in anemia, spasm or sclerosis of the retinal arteries, pigmentary degenerations, hemorrhages, thrombosis) or they may be due to actual inflammatory disease (such as seen in the simple or parenchymatous retinitis) or secondarily produced (such as seen in diabetes, nephritis, gout,

etc.). Rather generally throughout these states intestinal toxemia examinations should be engaged in. There have been eight cases of recurrent hemorrhages of the retina with more or less blindness markedly benefited, five of these being in instances of hypertensive nephritis.

SCOTOMA (CENTRAL, FALSE, TOXIC AND SCINTILLATING).

In false scotoma, toxic, and the scintillating forms, intestinal studies should always be made. Blind spots due to exudate of the retina are common in these chronic intestinal cases. Of such there have been at least 30 cases treated with most satisfactory results.

UVEITIS.

Sympathetic ophthalmitis and its subdivisions of uveitis serosa (iritis) and fibrinous iritis together with the accompaniments of choroidal change and papilloretinitis are commonly caused by intestinal toxemia. These serious troubles, helped only in small part by salicylate, atophan, or mercury treatments, have been judged as bound up in the mysteries of sympathetic causes when in the largest number of instances they are commonly primary or secondary to intestinal biological errors. In all of them, intestinal toxemia should be suspected, examined for, and treated.

VITREOUS CONDITIONS.

These are always interesting in an intestinal connection because of the common complaint of these people of "dancing spots" and "floating objects" in the vision. The condition of non-suppurative hyalitis, usually bound up with pathologic changes in the retina, ciliary body, choroid, or optic nerve, when not due to trauma, always deserves intestinal study and correction. Of the opacities in both the fixed and floating forms, the intestine often contains the etiological factors. In hemorrhages into the vitreous humor, even in the juvenile form, the bowel condition should be looked into. In synchysis scintillans these examinations are also in order.

CHAPTER XXII.

DERMATOLOGY.

Acne—Acne Rosacea—Dermatitis—Dermatitis Herpetiformis—Eczema—Erythema Exudativum Multiforme—Herpes Zoster—Lichen Ruber Planus—Molluscum Contagiosum—Nails—Pemphigus—Pityriasis Rosea—Pruritus—Psoriasis—Purpuras, The—Scleroderma—Seborrhea—Urticaria.

THE SKIN.

There is no specialty in medicine in which less is known of the causes of the disorders than in the domain of dermatology. Before this situation can improve in any worth-while way more data on the functions of the skin are required, because manifestly these are far more than is known, believed, or presented today. There are reasons to believe that the skin is an essential functioning organ of the body. More work on the physiology of the skin is urgently called for before there will be any uniformity of belief on many of the dermatological conditions.

The present status of dermatologists proves the widest variance of opinions. There is even marked difference of opinion on the very fundamentals of therapy. One group believes that dietetics is an important part of therapeutics, and just as prominent men believe this to be of no importance. Another grouping is seen in those who look upon most skin conditions as secondary to disorders within the body, and another that most of the factors of skin disorders are residential in the skin itself. When there is so much difference of opinion in the very fundamentals of the etiology and therapeutics of a specialty, one hesitates to present any ideas or opinions, especially when so little worth while is known concerning the physiology of the structures dealt with.

One may, however, record the fact that skin conditions as a class of disorders present a persistency and chronicity that is peculiar to themselves. These manifestly call for study of causative states and it is in these that intestinal toxemia un-

doubtedly plays an important part, mostly in the effects on the skin of soluble toxic products from the bowel in which skin disorders are produced by their elimination or to which skin tissues are peculiarly sensitive and react in certain eruptive ways.

Since it is not possible with our present-day limits of knowledge in medicine and chemistry to identify or estimate the presence or quantity of toxic substances in blood, it is, of course, still more impossible to figure how these affect the skin. Therefore, it is not possible to state whether these substances are primary exciting causes or contributory factors. That intestinal toxic states are important in the production of skin eruptions there is but little doubt in my experience and observation. In my opinion, they are the most important etiologic factors of chronic skin disorders, and states like increase of uric acid in the blood in many of them, as Schamberg has shown, should lead one's attention to the bowel as its cause, and not to uric acid alone as productive of skin states.

Among the microbic diseases of impetigo contagiosa, ecthyma and erysipelas, the various types of streptococci figure; in boils and carbuncles, follicular impetigo, sycosis and cocco-genic folliculitis, the staphylococcus; lupus vulgaris, tuberculosis verrucosa cutis, scrofuloderma and ulcus tuberculosum cutis, the tubercle bacillus; and probably organisms figure in large part in lichen scrofulosorum, tubercular erythema induratum, acne cachecticorum, papulo-necrotic tuberculides and sarcoid. Leprosy is due to the *Bacillus lepræ* of Hansen; anthrax to the *Bacillus anthracis*; glanders to *Bacillus mallei*; and rhinoscleroma to the pneumococcus. There are other microbic diseases, such as syphilis, and those due to vegetable fungi, like ringworm, favus, actinomycosis, blastomycosis, pityriasis alba, etc., and those due to animal parasites, scabies, pediculosis, etc. In many of these intestinal toxemia is an important contributing factor.

Since dermatological conditions, like those in other specialties, are largely affairs of internal medicine it is important that all matters pertaining to internal medicine are considered and that those findings pertaining to blood chemistry, blood counts,

basal metabolic estimations, the detection and quantitative estimation for the various metals in the urine should not be neglected.

ACNE.

In the various types of persistent acne, especially in acne vulgaris, bowel studies should be engaged in. As a rule the digestive disturbance and the anemia are secondary to these, and these are most important factors in the production of seborrhea. The same staphylococci in the intestines are found in practically all cases of staphylococcic skin conditions. It is probable here since staphylococci are denizens of the skin, that their presence in the intestine is more a matter of lack of immunity, and immunity production against staphylococci can be brought about by biological treatments rectally administered.

ACNE ROSACEA.

Bowel states may be of importance here. While no well-established case has been cured, marked benefit in several instances has been accomplished. Gastric conditions which may come from indiscretion in foods and fluids are commonly present. Sometimes these ensue in the stomach from toxic intestinal conditions.

DERMATITIS.

In the absence of a cause in the persistent or recurring case, intestinal studies should be made. I realize that there are many predisposing and exciting causes in this condition, but commonly none is as important as those originating in the intestine.

DERMATITIS HERPETIFORMIS.

In this disorder bowel studies should be made, although benefits from bowel handling are generally not observed in the skin.

ECZEMA.

Here is a line of disorders about which I am somewhat in doubt, although I feel quite sure that in the bowel are found

the most important factors by which to accomplish benefit. In the chronic forms of eczema, persistent for years, most remarkable results from bowel treatments were accomplished. It is no doubt true that in many of these cases several causes operate but one should not neglect the bowel condition, which may be most important to control to accomplish distinct benefit.

ERYTHEMA EXUDATIVUM MULTIFORME.

I have not seen a case of this yet that was not an instance of a distinct intestinal toxemia, this being also true of erythema iris, erythema nodosum, lupus erythematosus, scarlatiniform erythema, and acrodynia.

HERPES ZOSTER.

The idiopathic form of this disorder, especially that form causing extensive areas of necrosis, is no doubt toxic in nature, and one in which the bowel can figure in an important etiologic sense.

LICHEN RUBER PLANUS.

Intestinal studies should always be made in this condition.

MOLLUSCUM CONTAGIOSUM.

This disorder I believe is due to some direct skin infection, introduced from the surface.

NAILS.

Fragility, transverse furrowing, and striations of nails are commonly symptoms of these bowel conditions.

PEMPHIGUS.

Except in the malignant form, intestinal studies should be made in the vulgaris, foliaceous and vegetans types.

PITYRIASIS ROSEA.

Intestinal studies should be made in this condition.

PRURITUS.

What has been said of anal pruritus is true of pruritus in general. There have been not a few instances of more generalized itching, without manifest lesions, due to intestinal toxemia.

PSORIASIS.

No case of this disorder has been definitely benefited by the handling of an intestinal toxic condition. Several have shown benefit, but as a rule it has not been sustained.

PURPURAS, THE.

In purpura simplex, rheumatica and hemorrhagica, intestinal toxemia should be suspected and examined for. Other focal infections may exist as their cause.

SCLERODERMA.

In the circumscribed diffuse and in sclerodactylia a biologic and toxic bowel condition should be searched for, and corrected if found.

SEBORRHEA.

This disorder of the sebaceous glands commonly has an associated bowel condition. If the bowel condition has been standing for years and neglected, as a rule the condition, while it may be benefited, continues in lesser degrees.

URTICARIA.

Especially interesting in the recurrent types is the intestine as a main cause, the same being true of several types in the prurigos. Anaphylaxis from definite foods are of distinctly less importance in the causes than the bacteria in their effects on food proteins in the bowel. The instances of these cases with the relative erythemas, pruritus, and angioneurotic edemas have been many, and practically all have recovered. See Pruritus, Erythema and Angioneurotic Edema.

CHAPTER XXIII.

ORTHOPEDICS.

Chronic Arthritis, Atrophic and Hypertrophic Arthritis, Osteo-arthritis, Rheumatoid, Deforming and Spinal Arthritis—Myositis—Postural Defects—Various Joint Conditions.

CHRONIC ARTHRITIS, ATROPHIC AND HYPERTROPHIC ARTHRITIS, OSTEO-ARTHRITIS, RHEUMATOID, DEFORMING, AND SPINAL ARTHRITIS.

Taius Aurelianus in the fourth century A. D. considered arthritis as of gastro-intestinal origin with an hereditary pre-disposition. Paulus Ægineta regarded the intestinal tract with its disorders as contributing to both gout and arthritis. Popoff in 1887 experimentally produced arthritic lesions with streptococci. This has been confirmed by several others. Burbank and Hadjopoulos developed an active serum technic, and by careful choice of antigen from streptococci obtained from blood and upper respiratory sources carried the work further. Continuing with Hastings-Bedell strain of streptococcus and developing individual antigens from different types of streptococci obtained from various parts of the body, their work both diagnostically and therapeutically represents one of the real advances that has been made in the treatment of arthritis. There is little doubt that arthritis is not simply a disease of the joints, but a systemic one in which the joint manifestations are resulting or accompanying states.

The work of Burbank and Hadjopoulos is more concerned with foci remote from the intestinal tract, particularly from the various sources about the nose and throat. They, however, recognize that other foci of infection throughout the body can act as the initial cause, such foci being those that would come more particularly in the fields of the internists, otorhinolaryngologists, the gynecologists and genito-urinary specialists. It has not been recognized, however, that intes-

tinal organisms other than the streptococci can, by bringing on various toxic states in the blood, and, by their release into the blood from the intestines and their localization in various joints of the body, be the causes and no doubt often are distinct etiological factors of production. Such cases may be seen in the so-called rheumatoid arthritis, in osteo-arthritis, in the so-called atrophic and hypertrophic arthritis, and in spondylitis deformans, in which, while the streptococci isolated from regions "above the clavicle" can be the etiological factors, there are further resorption toxic states from the intestinal canal acting on these organisms in which the bowel state can be and often is the most important etiological factor.

In my work these arthritides are classified for clinical purposes into:

A. *Focal Infections*.—These are best illustrated in the acute arthritic conditions which follow or accompany acute tonsillitis, sinusitis, etc. When the arthritic condition has been long standing, often in them a focal infection may or may not be an important etiologic factor. The removal of the infection by operation commonly does not benefit the arthritis, yet often this is justifiable on the chance that it may. A focal infection could have been the original source and cause of the bone changes and its removal not benefit the arthritis, because:

1. The bone changes already present are permanent.
2. The removal of an original infection would not benefit the one now residential in the joints.
3. There may have been inaugurated other local factors not benefited by the removal of the focal source, and
4. Constant reinfection of the blood stream by causative organisms from the bowel, or symbiotic toxins from the same source. These are the commonest of all causes.

For some of these cases the methods of diagnosis and treatment of Burbank seem to be efficient.

B. *Intestinal toxemia and infections, of which there are many different kinds and types*.—The ileum and colon may be considered here as being the sources of focal infections in those instances where intestinal organisms have escaped from the

bowel confines (and this happens often without constitutional symptoms of any sort); and secondly, where the joint conditions are brought about by the constant resorption of bacterial toxic products, these bringing about changes in the anatomy of the ends of the long bones by destructive effects on the spongy tissues of the bones proximate to the joint and subsequently the cartilages and surrounding ligaments. The latter two depend upon normal anatomy of the spongy tissue for proper nutrition and the maintenance of normal physiology. Bacteria may clear from the blood, remaining only in the joint tissues and intestine, and the original ones may even clear from the joint tissues, and toxins from the same organisms in the intestines keep the joint process advancing. The gout cases belong to the resorption toxic group.

C. *The specific types of infections*, such as those due to the tubercle bacillus, gonococcus, *Spirochæta pallida*, *Bacillus typhosus*, a rheumatism organism perhaps, etc. These are not offered in this presentation.

D. *The arthritides of the menopausal woman*, which are probably endocrine in origin, and also not considered here, and

E. *A metabolic type, the cause or causes of which are not known*. Some of these, perhaps, are bound up in deficient states of elimination of the different body emunctories, some perhaps in excess calcium metabolism and accumulation, and no doubt there are other causes. This type of case, judged by the exclusion of the above forms, can and often does represent a factor which is brought about by the long continuation of the bacterial and toxic causes in which certain unknown changes of the general body metabolism have become established. These are also not presented here.

This article then deals entirely with the organism group, since, in my opinion, they represent the most important factor in the production of the chronic arthritides, are rarely diagnosed and treated properly, and the cases are commonly judged as instances of those in the other groupings. As the author desires to repeat, other mistakes made in the past are to judge them entirely as dietetic problems, or instances for physical treatments.

Burbank has shown that streptococci from the blood are often seen as diphthero-streptococci in morphology when isolated in the late fall and early winter. These are non-sugar fermenters and belong mostly to the *ignavus* type (Holman classification). Later in the winter months true streptococci types occur. In a recent communication¹ he has proven the presence of the *fæcalis*, the green streptococcus, the *infrequens* and the hemolytic. These were the most common, although the *mitis*, *salivarius*, *equinus* and the *subacidus* were also found. In the spring and summer months the cultures were negative.

The organisms isolated from human cases showed arthritic changes on animal experimentation with exudative changes, decalcification (sometimes with co-existent recalcification), proliferation and destruction of cartilage, epiphyseal changes and fibrous ankylosis. The bones showed on microscopical examination that the streptococci in the Haversian canals and canaliculi may be cultured from the bone and again produce the lesions on reinoculation. The muscle fibers frequently showed small hemorrhages with aniso-atrophic muscular involvements, the same as occur with humans, and also changes in the heart, liver and kidneys, commonly seen in the neglected and well-advanced case of chronic arthritis in the human being.

Cecil, Nicholls and Stainsby² have recently reported after painstaking observations that streptococci are isolatable from the blood in 61.5 per cent. of the cases and that in over 83 per cent. of these the organism is of the hemolytic type somewhat attenuated. Also that the same organism can sometimes be cultivated from the joints, these when injected into the blood stream of animals producing typical joint lesions (Burbank has also shown this) and they suggest that there is a specific strain. They express the opinion that arthritis deformans and chronic infectious arthritis are one and the same disease.

Since practically 90 per cent of all definite arthritis cases have the pathogenic streptococci isolatable from the intestinal tract, we now have confirmation of what I have contended for

¹ Burbank: Bull. Academy Medicine, N. Y., February, 1929.

² Cecil, Nicholls and Stainsby: Arch. Inter. Med., vol. xliii, No. 5, May, 1929.

years, namely that the intestinal canal is a potent focus of infection in these arthritic as well as many chronic disorders in man. In this connection, facts of symbiotic associations of the streptococci with other organisms in the intestinal canal should be emphasized since in these other organisms there may be far more significance than that belonging to the streptococci alone. The human intestinal canal is not suitable for growth of pyogenic streptococci unless other organisms make it so. Therefore to keep attention fastened only on the streptococcus (even if found in the joint tissues, and in my opinion the more particularly when it is) is only part of the significance both diagnostically and therapeutically. Commonly these associated organisms have far more importance etiologically and therapeutically than the streptococci alone, because if the symbiotic clique be not broken up, no matter what the treatment, the streptococcus infection is liable to continue.

Of course there are some rather straight streptococcus infections from the intestinal source in which the other organisms are not important, but they represent the more minor type of case of arthritis and more particularly the acute and subacute forms. Also a fact that must be mentioned is that other organisms than the streptococci can enter the circulation and cause the arthritic changes. Of such organisms there are many in the *Escherichia*, *Salmonella*, *Eberthella* and other enteroid groupings. As has been mentioned, also important in this connection is the fact that true chronic arthritic changes may be caused, maintained and advanced simply by the toxic substances from organisms residing in the bowel, these soluble toxins being resorbed into the blood over long periods of time bringing about the de- and hypercalcifying processes, atrophy, absorption, ankylosis, etc., without there being any streptococci or other organisms present in the joint or peri-articular tissues.

Therefore, while the streptococcus work of Burbank and Cecil *et al.* is most important, there is more to the subject than just streptococci in the stools, blood, joints or other focal sources, their identification and the use of strain vaccines and sera. The subject of etiology is much broader than this in

its significance regarding other organisms in the bowel, and therefore the whole therapeutic conception.

In 1920¹ I reported thirty-five cases handled by treatment of an intestinal toxemia. Since then this number has been added to, now making two hundred and five cases. All of these represented definite and generally well-established instances of chronic arthritis in which clinically no cause could be ascribed other than those of intestinal infections. In addition to these there were seventy-four cases of spinal arthritis with distinct limitation of mobility of the spine, and three hundred and eleven instances of spinal involvements not severe enough to cause anchoring of the vertebra (and of such cases there are far more instances than the profession has any idea of). In every instance of this group when streptococci, organisms belonging to the *Escherichia*, *Salmonella* and *Eberthella* were isolated from the blood, they were also found present in the intestinal canal and in not a few other focal infections as well (although the organism obtained from the extra-intestinal sources may more or less differ in significance).

In at least half of the instances the cases had been operated upon for the removal of focal infections or had been treated with various vaccines, sera, etc., as well as physical and electrical measures and various medications, mostly those containing iodine in some form given by mouth or intravenously. In at least half of the instances they were apparently a hopeless lot of patients with distinct advance of disease going on.

This is not the place to enter into a detailed description of each case or of the biologic handling in each, they all were more or less different, and none were handled in any other way biologically than is presented in the foregoing portions of this book. From this vast array of work certain facts stand out to me in the results accomplished, which after all are the most important to record. Whether focal infections are present in the head sinus or throat or not, present anywhere else in the body or not, the biology of the intestinal

¹ Bassler: Amer. Jour. Med. Science, vol. clx, No. 3, No. 583, p. 351.

canal in the chronic case should be fundamental in interest in the etiology and therapeutics, although focal infections elsewhere often deserve attention. Whatever organism or organisms are found in extra-enteric locations or in the blood or joints, the intestine should receive primary attention on account of being the continued location and the source of keeping up the infection or causing the reinfections which more or less persist in many of these cases.

In the clearing up of the streptococci in the blood and joints, the streptococci must first be cleared from the gut, and this is more a matter of breaking up the symbiotic cliques in which they occur than the use of streptococcic vaccines or sera introduced through the skin, although these may be indicated at times. With streptococci in the blood and joints, they are always present in the bowel, and, even though they are culturing in the blood and bone tissue, they are doing so largely because of resorbed toxic material from the bowel from organisms which are symbiotic with the streptococci, thus keeping up the infection in the intestine as well as outside in the body.

With the other organisms that can cause the conditions, as well as the streptococci, the importance of this association of infections holds true, and it holds true not only in these conditions, but in all systemic infections of chronic nature. It is this fact insofar as streptococci are concerned that has largely brought the discouragement in handling streptococcic infections by streptococcic vaccines and sera alone. Streptococcic infections are best controlled by controlling the associations in which they occur and not by direct attack on the streptococci themselves.

There are many cases of these joint conditions in which the blood and joints are bacteria-negative even on repeated examinations. Of course the organisms may still be present and not isolatable or culturable. Also they may have been present and now gone, the other organisms mentioned being the continuation cause, or the toxins from the bowel are continuing the mischief, since there is little doubt that resorbed toxins and the metabolic toxic responses to them can cause these lesions

even to extreme degrees. There is no doubt that the largest factor of significance in these conditions is one of infection, and in this those of the bowel are of primary importance. In the handling of this disorder the methods should be directed more to the intestine than is now believed important. It is not a dieting affair fundamentally, as Pemberton and others believe, excepting insofar as dieting is indicated according to the biology of the intestines. To diet directly on a diagnosis of an arthritis is a type of medicine that should be only of historic interest. As a rule the atrophic type is almost always an infective form, while the hypertrophic may be, or is in part or totally, more metabolic in classification.

So far as other measures of treatment are concerned the time for physical measures is about a year after the infection is controlled, practically never when the infection is present. Whatever the kind of physical treatment in a well-established case, to depend upon it alone and neglect the infection is largely losing valuable time and money, and accomplishing no worthwhile benefit. Benefits come at times with the various iodine or benzoic preparations, especially when used intravenously, sometimes with low calcium diets and occasionally with climatic and tonic measures. Radium has been of little value in my cases. The menopause case may require endocrine treatment as assistant. Rest, restraining splints, heat, with aspirin or atophan have been used to accomplish benefit along the way.

MYOSITIS.

In the vague types of this condition the possibility of a toxic and infected intestinal state should be thought of.

POSTURAL DEFECTS.

The question of reconstructing chronic patients along only physical lines, as Goldthwait has suggested, is worth while as far as it goes. There is no doubt that deranged anatomy and physiology are not only a physical matter. In visceroptosis, intestinal stasis, diseases of heart, lungs, nervous system, arthritis, etc., the biology of the intestinal canal also figures prominently as a contributing factor. Poor metabolism, faulty

organ function and physiology, invalidating factors, all are ensuing states from chronic intestinal toxemia. In breaking up a long vicious chain in which posture defect figures, the biology of the bowel is an essential part of organ function and physical and vital strength. While the correction of posture defects in many people is a necessary education for them to have and maintain in practice, the best results seen are those who at the same time have perfectly healthy bodies bacteriologically and chemically.

VARIOUS JOINT CONDITIONS.

It is not necessary to detail a long series of conditions (always more or less vague in any entity sense) that occur in joints. What has been stated in arthritis covers some, but there are others in which this term may not be properly accurate so far as we know. Instances of easy subluxation, "graty knees" or shoulder joints, persistent types of pain, and distress in joints, etc., not accompanied by bone changes, limitations of motion, etc., are not uncommonly instances in which intestinal studies may be wise to engage in. Sometimes these involve muscles, various nerves, peri-articular tissues, ligaments, etc., and since they are so commonly met with in practice, unless explained or easily corrected in simple ways, they may justify intestinal study for their possible cause and an important contributing factor for their correction. In these, of course, are included the various types of myofacitis already presented in the chapter on Internal Medicine.

CHAPTER XXIV.

UROLOGY.

Acute Suppuration of the Kidney—Calculus, Renal, Ureteral, and Urinary Bladder—Chronic Pyelonephritis—Cystitis—Perinephritis—Prostatitis—Pyonephrosis—Renal Tuberculosis—Ureteritis and Ureteral Stricture—Urethritis—Vesiculitis.

Where do the organisms which infect the urinary tract come from? With the one exception of the tubercle bacillus they are all intestinal in origin. Thomas R. Brown¹ studied the infections in one hundred cases from Dr. Kelly's private clinic and found the following in sixty-seven cases as reported in the old classification: *B. coli communis* (present in one-half of all); *Staphylococcus pyogenes, albus*; *Staphylococcus pyogenes, aureus*; *B. proteus vulgaris*; *B. pyocyaneus*; *B. typhosus*; *B. tuberculosis*. In twenty cases of pyelitis the infections found were: *B. coli communis*, 7; *B. tuberculosis*, 6; *B. proteus vulgaris*, 4; *Staphylococcus albus*, 2; one was omitted.

The works of several other authors on urology^{2 3 4 5} duplicate the above organisms and also add the following: Cocci (undoubtedly the intestinal forms); *B. alcaligenes*; *B. pyocyaneus*; *Streptococci*; *B. welchii*; *B. lactis aërogenes*.

If at the time these studies were made advanced cultural methods and knowledge of those known today had been engaged in, it could be proven that practically in every instance of kidney, ureter and bladder infections they were due to some organism, and that all, with the exception of the *B. tuberculosis*, are intestinal in origin. It may therefore be stated definitely that practically every case of an urinary infection, whatever treatment is engaged in by the urologist, requires also biologic study of the intestinal canal, even after operation in the tubercular kidney cases. One must not forget that, with the urinary tract as with the lungs, tubercle bacilli are in themselves but

¹ Brown, Thomas R.: Johns Hopkins Hosp. Rep., xi, 89, 1905.

² Suter: Ztsch. f. Urol., 1, 327.

³ Cheetwood: Practice of Urology and Syphilology, 4th Ed., p. 656.

⁴ Lowsley and Kirwin: Text Book on Urology, p. 485.

⁵ Kelly and Burnam: Diseases of Kidneys, Ureters and Bladder.

poor types of infecting organisms and that the ravages of tubercular infections are accomplished by the mixture (symbioses) of the *B. tuberculosis* with other organisms.

There are no more hopeless and persistent types of infection than those found in the urinary tract. Most indirect and far removed as regards immunity in the body, these extra-peritoneal structures are still close in location to the intestines. More than that they undoubtedly are eliminating structures, and it is very probable that the kidneys are provided with abilities to eliminate organisms from the blood stream as well as the chemical urinary constituents and fluid. Peculiarly, then, any organisms in the blood stream, or picked up by the lymphatics from the peritoneum, are most liable to reach the kidneys. If an infection continues in the intestine, once the urinary tract becomes infected, constant reinfection is most liable to occur, and this is one of the difficulties of controlling these infections, as handled today, and the main reason for their persistency. As a rule, however, if the intestinal infection is controlled, that of the urinary tract often responds to treatment in reasonable ways.

Bacteria are often found near the meatus, the deeper portions of the anterior urethra generally being sterile. The organisms most often found are the staphylococcus, sarcina, *Escherichia coli*, *Proteus vulgaris*, streptococci and various of the intestinal cocci. As reported by David¹ organisms found in infected urinary organs were the following:

<i>Aërobes.</i>	<i>Anaërobes.</i>
23 <i>Bacillus coli</i> .	4 Black-pigment-producing bacilli.
15 <i>Staphylococcus albus</i> .	4 Gram-negative, influenza-like bacilli.
3 <i>Staphylococcus aureus</i> .	2 <i>Staphylococcus parvulus</i> .
5 <i>Bacillus enteritidis</i> .	1 Gram-negative coccus.
3 <i>Bacillus fæcalis alcaligenes</i> .	1 <i>Bacillus funduliformis</i> .
2 <i>Bacillus proteus</i> .	2 Gram-positive staphylococcus.
2 <i>Bacillus pyocyaneus</i> .	
1 <i>Streptococcus</i> .	
1 <i>Bacillus pseudodiphtheriæ</i> .	
1 Unidentified Gram-positive diplococcus.	
1 <i>Pneumococcus</i> .	
1 Influenza-like bacillus.	

¹ David: *Gynec. and Obst.*, xviii, 432, April, 1914.

Infections of the renal pelvis practically always result from bacteria of the *Escherichia* group, those of the bladder being more particularly the pyogenic organisms, also from the bowel. In the presence of accessory organisms which cause the splitting of urea into ammonia and water by mixed infections such as the proteus, the pyogenic cocci, etc., the more particularly infecting organisms may engraft themselves with the production of lesion.

There are various routes of invasion of the genito-urinary canal, the most important being the descending or excretory route in which infection reaches the kidney substance from the blood, it being proven that living bacteria may enter the urine from a kidney clinically sound. It has been suggested that the cells of normal kidneys cannot transmit a living bacterium, but this is not true. It is probable that in the majority of instances single strain bacteria presented to kidney tissue would pass through the tissue without the production of lesion. But in the presence of other organisms, and these are commonly associated with definite infective types, infection may ensue and lesions take place. In the instance of these the infection is almost always bilateral, although it may be distinctly milder in one kidney than in the other.

As Crabtree has suggested, pyogenic cocci reaching the kidney through the circulation set up an infection in the glomeruli while the *Escherichia coli* are more likely to pass through the cortex substance and attack the tubules. Hence the more common finding of the *Escherichia coli* infecting the pelvis of the kidney while the coccal infections attack the perinephritic tissue. Of course such bacteria may be derived from any part of the body, such as the roots of teeth, tonsils, adenoids, etc., but in the majority of instances they are direct from the intestines.

Invasion of the bladder and kidney can take place as an ascending infection following instrumentation or by ascending urethral infection, and infections may occur by the lymphatic route or by direct invasion, by trauma, or rupture of a neighboring abscess. However, there is little doubt that the majority of urological infections are central in origin, namely de-

scending from the kidney, this of course excepting the venereal types in which entrance is by way of the meatus. There are also in urological conditions accessory causes such as the retention urethral or ureteral conditions, trauma, toxic influences, reflex states, etc.

The *Escherichia* group practically comprise 90 per cent. of the infections of the pelvis of the kidney, while in those of the bladder the pyogenic and the *Escherichia* group are about even in number. The most important thing pertaining to all of the infections of the internal urinary structures is the incidence of mixed infections, and in the production of these, practically all of the intestinal organisms can operate. Of importance, too, in certain single strains or simple mixture types of infection, are the toxic substances in the blood derived from bacteria still in the intestinal canal which act favorably to an infection that has reached the kidney structure and thus result in the production of lesions in that organ, the ureters, the bladder, and the base bladder structures in the male. It is a well-known fact that the control of infections in the kidney or bladder is a bane of the urologist's work, and urological infections will never be understood within the compass of intelligent control as long as attention is paid to only a few organisms found locally in the infective locations. The associations must particularly be studied and also the biologic conditions in the intestinal canal producing toxic substances which act symbiotically with the infection already in the urinary tract.

Another very important point for the urologist to remember is that in the presence of an intestinal infection it is almost impossible to clean up a urinary infection on account of constant reinfection or toxicity effects from the bowel through the blood stream to the kidney and bladder tissues. Until these facts are incorporated in the study of urological infections, control of these infections will not be accomplished to substantial degrees.

ACUTE SUPPURATION OF THE KIDNEY.

By this we understand a condition of multiple suppurating points in both kidneys, generally abscesses chiefly in the cortex

substance. One usually has little chance to study the infection in these cases, for their clinical course is, as a rule, rapid and fatal. Acute suppurative nephritis is usually an accompanying condition with pyemia or septicemia in which an acute suppurative bilateral nephritis is co-incident and subordinate to general infection. There is, however, an occasional instance of a mild type of case in which the biologic study of the intestinal canal may be important. In septic infarcts the lesion usually takes on the semblance of a severe acute pyelonephritis and in this instance the infection is generally that of cocci, and those in which the invasion is not by way of the skin or plainly evident focal infections may necessitate intestinal study.

CALCULUS, RENAL AND URETERAL, URINARY BLADDER.

In these, even when accompanied by retention, ulceration, or inflammation, intestinal studies are often indicated. As a rule, however, the control of the intestinal infection has no appreciable effect when stones are already formed; particularly is this true in the bladder types. However, the very presence of stones anywhere in the canal is enough indication for intestinal studies to be made, because this may be important to prevent or assist in preventing re-formation of stones after their removal. Where there has been recurrent formation of stones the rule is that there is so much infection of the organ in which they occur that clearing up in the intestinal canal brings no curative or totally preventive benefit.

CHRONIC PYELONEPHRITIS.

These cases are met with in all grades of severity, and in the mild ones or those of long duration, intestinal studies may be called for, although usually when the condition is distinctly present, the kidney tissue goes on to distinct degeneration and destruction.

CYSTITIS.

There are non-bacterial types of cystitis. The important ones, however, are the bacterial forms in which the acute,

chronic, interstitial, and pericystitic forms are of interest. As a rule, acute cystitis does not require extensive intestinal studies except in instances of frequent recurrence, but in the chronic, ulcerative and pericystic types intestinal studies are essential. The intestinal canal can act as a source of constant reinfection of the urinary bladder. This may come from certain parasitic types of *Escherichia coli* and the pathogenic cocci, or, what is more important, these associated with other intestinal organisms.

PERINEPHRITIS.

In fibrolipomatous perinephritis and in the suppurative forms it may be wise to engage in intestinal studies. In pyelitis granulosa, malacoplakia, pyelitis cystica, and in calculous incrustation, intestinal studies are usually in order.

PROSTATITIS.

This is usually accompanied by seminal vesiculitis and since the prostate and seminal vesicles may become infected from the blood stream route without the infection being passed to these structures by way of the urinary tract, intestinal studies may be called for. There have been several instances of chronic prostatitis in which the posterior urethra and the seminal vesicles were involved wherein the control of the intestinal infection plus local treatments by a urologist accomplished most satisfactory results. These of course were non-gonococcal and tubercle bacilli infections.

PYONEPHROSIS.

Even in the instance of distinct retention factors intestinal studies should be made in these cases. In those more chronic in type either ensuing from a mild infection in a dilated kidney, or simply the end phase of severe chronic pyelonephritis, the gravity of the case and the possibilities of benefit by correcting intestinal errors gauge whether intestinal work should be done or not.

RENAL TUBERCULOSIS.

In the case of renal tuberculosis, as a rule, but little can be done medically. There are instances, however, of single kidney infection and tubercular lesions of the bladder in which it might be wise to engage in intestinal studies, because the tubercle bacillus in its ability to destroy tissue is very largely dependent upon a mixed infection, and since most of the infections of the urinary canal are intestinal in origin, such studies may be called for or deemed wise in some instances.

URETERITIS AND URETERAL STRICTURE.

In occasional instances of these, intestinal studies are called for as an assisting therapeutic factor of benefit in ureteral spasm, stricture, and even in ureteral kinks. As a rule when definite change from normal exists, no anatomical benefit can be accomplished.

URETHRITIS.

There are forms of urethritis, non-gonorrheal or tubercular, in which the intestinal canal may figure as a cause. More particularly, however, this is important in the posterior urethritis types, and those in which the prostate and seminal vesicles are also bound up in the processes of infection.

VESICULITIS.

What has been stated regarding prostatitis is true here.

CHAPTER XXV.

GYNECOLOGY.

Cervicitis, Endocervicitis and Metritis—Cystitis (Pyelitis)—Infectious Oöphoritis—Parametritis and Pelvic Cellulitis—Pelvic Abscess—Pelvic Adhesions—Postoperative Ileus—Pruritus Vulvæ—Puerperal Sepsis—Salpingitis.

The physiology of the uterus and ovaries is considerably dependent upon general states. One needs but to collate the cessation of menstruation in infections, metabolic and glandular disturbances to realize that menstrual function is considerably dependent upon general states of health. In this sense chronic intestinal toxemias may be an important factor in this or that case because these bowel conditions are essentially a continued infection.

Many times gynecologists are called upon to treat conditions bound up in disorders of neighboring organs such as those of the urinary bladder, ureters and kidneys, stomach and intestine, sigmoid and rectum, gall-bladder, peritoneum and omentum, appendix, etc., and as these are produced originally from bowel infections, intestinal studies are significant. The gonococci are not denizens of the intestinal canal and infections of this organism in the vagina, urethra, Skene's and Bartholin's glands, cervix, endometrium and tubes are local infections. One must realize, however, that gonococcic infections are largely self-limited in time and commonly become mixed or ultimately become subinfected with intestinal organisms and in this sense the intestinal canal may have significance. Especially is this true in some of the chronic types of inflammations in which the pelvic tissue is involved. When distinct erosion is present one of the operations for its removal is required. Intestinal treatments will not cure it. Its presence should only be taken as suggestive of an intestinal condition.

CERVICITIS, ENDOCERVICITIS AND METRITIS.

In the so-called non-septic forms of these conditions, intestinal states are commonly the causes by which the conditions are brought about by way of transmitted infections from the bowel, in which instances the organisms rather quickly disappear from the tissues and the local condition is kept up by absorptions of irritating substances from the lower ileum and colon. One must of course keep in mind how readily infection of the cervix and uterus can occur by way of the vagina. But very few of these, other than gonorrhea, are capable of persistency and marked effects on these tissues without assistance from the bowel or altered anatomy. Any local traumatic lesion like laceration of the cervix due to childbirth, added to by infective inflammatory process or bacterial lytic products from the bowel, would make the etiological factors more complete.

Infective endometritis most commonly as a result of puerperal sepsis is due largely to organisms intestinal in origin. Under normal conditions the uterus is more or less in steady contact with intestinal organisms, which in puerperal states may easily cause local sepsis. It is probable that chronic interstitial endometritis is in some way a factor in this connection. In the soft large boggy uterus, look to the intestines for a possible cause.

CYSTITIS (PYELITIS).

The cystitis of women is generally from intestinal organisms, introduced rarely by the urethra but most often by the blood stream route by way of the kidneys. In all instances of recurring cystitis or infective types of urine in the female, biological bowel studies are called for. The same is true of pyelitis, even the postoperative form, and especially in the chronic form.

INFECTIOUS OOPHORITIS.

Since this is mostly due to streptococci of intestinal origin, whether following peripheral sepsis or not, the bowel should be thought of.

PARAMETRITIS AND PELVIC CELLULITIS.

Since the pelvic cellular tissue has low resistance and is one through which infection easily spreads, in the absence of evident local causes and sometimes in the presence of them, the bowel is a common source of infection and reinfection. These infections, however, may come by way of the urinary bladder, anal skin, perineum and vagina.

PELVIC ABSCESS.

This is most often due to infections mixed with or following gonococci, the infection with pus formation occurring in the tube, ovary, or cellular tissue. Intestinal studies are called for after surgery.

PELVIC ADHESIONS.

These, of course, commonly occur from infection of the tubes. The condition, however, occurs often when disease of the tubes is not present. Those which take place without definite initial onset or cause, and those instances met with at operation, require intestinal studies for their explanation and perhaps their control.

POSTOPERATIVE ILEUS.

Bound up in part with the instances of this condition are the chemical conditions in the bowel which had been there usually for long periods before operation and neglected, and which with shock and the taking of an anesthetic, suddenly precipitate the condition. The status ensuing is that of parietic stasis of stomach or small intestine, generally of both, in which the chemistry in the bowel is practically that of obstruction with its consequent and overwhelming toxemia. While I would not go as far as to advise biological studies of the bowel in all instances of intended surgery, and certainly not in the presence of this condition to waste any time in fussing with these diagnoses, I believe that after it has occurred and recovery has taken place, the condition should be taken as an

index of possible bowel disorder and examinations should be made.

PRURITUS VULVÆ.

See Pruritus (Anal).

PUERPERAL SEPSIS.

This condition may occur from poor aseptic technic, or meddlesome and unnecessary digital examinations; but when these have been conducted properly, the accoucheur should not become conscience stricken when his case becomes septic after childbirth. With the most precise technic and care in handling all the way through parturition, infection can and does occur with intestinal organisms present in the uterine and periuterine tissues at the time of delivery. In non-pregnant states the organisms would perhaps do no special harm, but under the conditions of the gravid uterus being emptied they can cause sepsis. For this reason, and when it has happened, streptococcic gut infections particularly should be known before delivery or cleaned up afterward. If this is kept in mind, a considerable part of the maternal death rate after delivery could be controlled.

SALPINGITIS.

The acute form of this condition is probably bound up more importantly with gonorrheic infections, although even here intestinal organisms may figure in the process. The normal tube, especially at its middle, in its mucous membrane villus formation suggests a perfect nidus for infective inflammation, and this tube is in direct contact with the blood-vessels, commonly infected with low virulent types of organisms from the intestine, and which may gain lodgment in the tube. In chronic pelvic inflammation, even as a result of gonorrheal salpingitis, mixed infection is an important factor. These conditions, of course, are mostly of surgical interest, but in the conservative handling infective intestinal states may be important factors for control.

CHAPTER XXVI.

PEDIATRICS.

Acidosis and Alkalosis—Acrodynia—Allergies—Anorexia—Celiac Disease—Dilatation of the Stomach—Gastralgia—Gastritis, Acute and Chronic—Intestinal Colic—Malnutrition—Rheumatism—Sinuses—Tonsils—Vomiting.

ACIDOSIS AND ALKALOSIS.

This has already been discussed in the section on Internal Medicine. One may say here in repetition that in children, as it is in adults, a tremendous amount of confusion exists. In distinct acidosis and alkalosis, in ketosis and acetonuria, intestinal studies when properly done usually give the solution of the clinical problem. It must be remembered throughout all of pediatrics that the majority of the infections of the intestinal canal met with in the adult originated in the first few years of child life, and, no immunities having been acquired, the infection persists, taking a number of years to cause clinical symptoms.

It is most common in taking history on the toxic case of an adult to disclose the mysterious or ill-understood condition that was present in the childhood years of the patient. Sometimes this is diarrheic, it may have been a long course of constipation, a fevered state of unknown nature, a digestive disturbance, or some condition which has been designated under the entity of some of the terms still used in pediatrics, and which are in no sense a true clinical entity. For this reason it is important that interest in biologic states of the infant and child should be awakened among pediatricists, because if one is interested in prophylactic medicine there is no doubt that the time to correct, control, or benefit intestinal toxemia in many instances is during the early days of the toxemia, namely in infancy and childhood.

ACRODYNIA.

In these instances intestinal studies should be made.

ALLERGIES.

It has already been mentioned in several parts of this volume that, in addition to the effects of specific proteins upon susceptible individuals, the biologic chemistry of the intestinal canal is the most important factor in the understanding of allergy in a general way. There are in children definite instances of allergic states such as hypersensitiveness to cow's milk, etc., but wherever it is suspected that a condition or an illness is represented in an allergic phenomenon, intestinal studies are called for. Commonly an individual would not be sensitive to a protein if the biology in the intestine did not make him so in the production of certain amino-acids and toxic substances, which toxic bodies would not occur from like proteins if the bowel biochemistry were normal.

ANOREXIA.

This is also a symptom common to a moderate degree in a great many diseased states, and is oftenest of brief duration. Where it is a feature in the case and no cause can be ascribed, intestinal studies may be called for.

CELIAC DISEASE.

Celiac infantilism, Herter's infantilism, intestinal infantilism, etc., is a chronic intestinal indigestion met with in artificially fed infants and children. It is assumed to be due to the *Bacillus bifidus*, *Bacillus infantilis*, *Micrococcus ovalis*, parasitic forms of *Escherichia coli* and other proteolytic bacteria. The carbohydrate intolerance, increased fat content and diarrhea are due to an infection. The stools should be biologically studied; however, because of the youth of the subject (young subjects are not good material for biologic work) the best treatment still remains in lactic acid milk, corn syrup, fruits, specially prepared carbohydrates, gentle purgation, etc.

DILATATION OF THE STOMACH.

In these instances intestinal toxemia being the commonest cause, it should be kept in mind.

GASTRALGIA.

Like vomiting, gastralgia is a symptom of various conditions, and these various states need not be gone into here. However, it may be produced by an intestinal toxemia.

GASTRITIS, ACUTE AND CHRONIC.

Usually in the acute forms a dietetic indiscretion explains its reason, but in the chronic type of gastritis an intestinal toxemia can be an exciting cause.

INTESTINAL COLIC.

One of the briefs I have against the modern-day pediatrics is that whenever there is a digestive disturbance in a child or infant, indiscretion of diet or improper feeding is immediately ascribed as its cause. In tympanitis, umbilical colic and intestinal indigestion; in the diarrheic disturbances; acute and chronic ileocolitis; constipation; chronic intestinal indigestion (especially in older children); dilatation of the colon, and others, biologic intestinal studies have far more importance than modern-day pediatrics ascribes to them. Especially is this true in instances of ileocolitic ulceration; chronic diarrheic states; appendicitis; conditions of the liver; non-tubercular diseases of the peritoneum; laryngeal and bronchial conditions and lung inflammations; disorders of the heart and urinary apparatus; epilepsy and headaches; dreaming, nightmares, somnambulism, hysteria, mental deficiency; the muscular atrophies and dystrophies; neuritis; morbid conditions of bone, blood and skin states; the nervous and precocious child, and here and there throughout quite a list of disorders described as pertaining to adults in books of internal medicine and the specialties. It must be remembered that men and women are merely grown-up boys and girls, and many of the diseases and conditions of the adult

are present likewise in the young, and commonly persist from childhood on to adolescence.

MALNUTRITION.

Malnutrition is more particularly due to improper feeding or unhygienic conditions. There are instances seen quite often in a child wherein disturbances in the gastro-intestinal canal, especially those of intestinal toxic states, are undoubtedly a factor. These are seen more particularly in the chronic types of malnutrition where no other cause for malnutrition is evident.

RHEUMATISM.

While the definite exciting cause for this condition is still not known there is reason to believe from its symptoms that it is an infection, and far more often an infection from the intestinal canal than from tonsils, sinuses, roots of teeth, etc. In the chronic articular types as well as in the erythema of various forms in the tonsillar or pharynx types, and in the rheumatic muscular conditions, intestinal studies may be called for. See section on Internal Medicine.

SINUSES.

See Otorhinolaryngology.

TONSILS.

See Otorhinolaryngology.

VOMITING.

Vomiting is a symptom of so many other conditions and infections common in infancy and childhood, that a review of its causes and varieties is not in order here. There is a toxic type of vomiting in which intestinal studies may be called for, seen most often in the recurrent or cyclic cases.

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